ASSESSMENT OF NUTRITIONAL STATUS AND ANEMIA IN CHILDREN WITH CONGENITAL HEART DISEASE -A CROSS SECTIONAL STUDY

Dissertation

Submitted to

THE TAMILNADU Dr. M.G.R MEDICAL UNIVERSITY

In partial fulfilment of the requirements for

the award of the degree of

M.D. PEDIATRICS

BRANCH VII

MAY 2019

CERTIFICATE I

This is to certify that this dissertation entitled **"Assessment of Nutritional Status and Anemia in Children with Congenital Heart Disease"** is a bonafide record of the work done by **Dr. Nargis Rabiya A.N.** under guidance and supervision of **Dr. Elizabeth K.E.** in the Department of Pediatrics during the period of her postgraduate study for **M.D. Paediatrics [Branch-VII]** from 2016-2019.

Dr. Elizabeth K.E., MD
[Guide]
Professor and HOD
Department of Paediatrics,
Sree Mookambika Institute of
Medical Sciences [SMIMS]
Kulasekharam, K.K District,
Tamil Nadu - 629161

Dr. Rema V. Nair, M.D., D.G.O.,
Director
Sree Mookambika Institute of
Medical Sciences [SMIMS]
Kulasekharam, K.K District,
Tamil Nadu -629161

URKUND

Urkund Analysis Result

Analysed Document: NARGIS PLAGERISM.pdf (D42493832)
Submitted: 10/12/2018 7:44:00 PM
Submitted By: n.rabiya7@gmail.com
Significance: 4 %

Sources included in the report:

final compile thesis for antiplegiarism.docx (D30896235)
CH Case 2.docx (D19474944)
https://www.cdc.gov/ncbddd/heartdefects/facts.html
http://www.scielo.br/scielo.php?script=sci_arttext&pid=S0104-11692012000600003
https://myhealth.alberta.ca/Health/Pages/conditions.aspx?hwid=tx4010
http://www.cpho.or.kr/journal/download_pdf.php?doi=10.15264/cpho.2015.22.1.54

Instances where selected sources appear:

19

CERTIFICATE II

This is to certify that this dissertation work titled **"Assessment of Nutritional Status and Anemia in Children with Congenital Heart Disease"** of the candidate **Dr. Nargis Rabiya A.N.,** with registration Number 201617701 for the award of **MD** in the branch of **Pediatrics [Branch-VII]**. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **4** percentage of plagiarism in the dissertation.

Guide & Supervisor sign with Seal.

DECLARATION

In the following pages is presented a consolidated report of the study **"Assessment of Nutritional Status and Anemia in Children with Congenital Heart Disease"** on cases studied and followed up by me at Sree Mookambika Institute of Medical Sciences, Kulasekharam from 2016-2019. This thesis is submitted to the Dr. M.G.R. Medical University, Chennai in partial fulfilment of the rules and regulations for the award of MD Degree examination in Pediatrics.

Dr. Nargis Rabiya A.N

Junior Resident
Department of Pediatrics,
Sree Mookambika Institute of Medical Sciences,
Kulasekharam, Kanyakumari District.
Tamil Nadu 629161.

ACKNOWLEDGEMENT

It's time to look back and express my gratitude to all those wonderful people without whom my journey as a postgraduate wouldn't have been a smooth sailing, as smooth and as beautiful as it was. Hence at the outset let me thank the almighty for giving me all I have and making all this possible.

I consider it my distinct privilege and honour to have worked under the guidance and supervision of ***Dr. ELIZABETH K.E****, professor and HOD of Pediatrics, Sree Mookambika Institute of Medical Sciences, Kulasekharam. I express my feeling of gratitude and sincere appreciation for her guidance, constant support and encouragement in making the study possible. Her constant help, patience and guidance helped me at every stage from conception to completion of this dissertation. I am blessed and privileged to be taught by such an eminent teacher.*

My deepest gratitude and respect to my beloved Chairman ***Dr. C.K. Velayuthan Nair*** *and Director* ***Dr. Rema V. Nair*** *for providing facilities to accomplish my dissertation work.*

I extend my sincere heartfelt thanks to Professor ***Dr. C. Sulekha,*** *former HOD, whom I am highly indebted for her relentless help, proficient ideas and constant encouragement.*

I express my gratitude to the ***Dr. Rukmini K,*** *Professor, for her invaluable help in preparing this dissertation.*

I express my gratitude to the ***Dr. Nedunchezhiyan,*** *Associate Professor, for his invaluable help in preparing this dissertation.*

I also express my deep sense of gratitude and sincere thanks to ***Dr. Sanjay Masaraddi****, Associate Professor, for his immense help, unconditional support, valuable suggestion and encouragement in completing this study*

I express sincere gradtitude to my co postgraduates to Dr. Thirumalai Vasan, Dr.Mohanaselvan for helping and supporting me in preparing this thesis

I am thankful to all other non-teaching staff members for their support and co-operation in completing this work.

Lastly, I am forever indebted to my parents and my siblings for their support and understanding throughout my study period.

I would like to thank Leos Data Makers for their immense help in completing this dissertation.

I am grateful to all the parents of children of the study for their kindly cooperation.

Finally, I pay my prayers to the Almighty God for his blessings without which I could not have completed my dissertation.

ABBREVIATION

CHD	Congenital Heart Disease
ACHD	Acyanotic Congenital Heart Disease
CCHD	Cyanotic Congenital Heart Disease
ASD	Atrial Septa Defect
VSD	Ventricular Septal Defect
PDA	Patent Ductus Arteriosus
TOF	Tetratolgy Of Fallot
IYCF	Infant And Young Child Feeding Practices
WfA	Weight For Age
HfA	Height For Age
WfH	Weight for Height
BMI	Body Mass Index
CED	Chronic Energy Defeciency
MUAC	Mid Upper Arm Circumference
Hb	Hemoglobin
MCV	Mean Corpuscular Volume
MCH	Mean CorpuscularHemoglobin
MCHC	Mean Corpuscular Hemoglobin Content

ABSTRACT

INTRODUCTION:

Congenital heart defect (CHD) is the most common defect among all birth defects representing a major global health problem. Twenty-eight percent of all major congenital anomalies comprise of heart defects. Congenital heart disease (Cyanotic and Acyanotic) occurs in approximately 0.8% of live births.In India, the prevalence of CHD is not uniform across the country and varies from 0.8 to 5.2/1000 patients in community-based studies while the prevalence ranges between 3.9 and 26.4/1000 live births in hospital-based studies in India, which is not uniform across the country. The burden of CHD is high in developing countries like India, due to the high birth rate and critical nature of CHD requiring expensive surgical and non-surgical interventions. CHD is considered a real challenge because of the complex interplay between medical, surgical, dietetic and socio-economic factors.

AIMS AND OBJECTIVES:

- To find out the Nutritional Status in children (1-12years) with congenital heart disease by using anthropometric measurements, clinical assessment and dietary evaluation
- To assess degree and type of Anemia in children with congenital heart disease using assessment of hemoglobin, red cell indices, red cell distribution width and peripheral smear

METHODS:

We conducted an cross sectional study of 80 children aged 1-12 years of age with congenital heart disease chosen by purposive sampling technique,attending as

Outpatient and Inpatient in the Department of Peadiatrics, Sree Mookambika Institute of Medical Sciences, Kulasekharam. over a period of 18 months

RESULTS:

In this study on 80 children, 63.8 % were in the age group 1-12 years whereas 36.2% belonged to age group 12mon-59mon.The male to female ratio was 1:1. 81.3% had ACHD;out of which VSD was the most common(35%).18.7% had CCHD,out of which TOF was the most common(13.7%).69.2% of ACHD were underweight in comparison to 35.7% in CCHD.with significant p value 0.025,42.9% of ACHD were stunted in comparison to 57.1% in CCHD with significant p value of 0.004,80% of ACHD were wasted in comparion to 20% wasted in CCHD. 21.3% of ACHD had anemia 17.5% of CCHD had polycythemia.21.2% had decreased red cell indices indicating microcytic hypochromic and 36.2% had increased RDW with p value 0.000 indicating nutritional anemia.According to Peripheral smear,16.2% had microcytic hypochromic anemia.

CONCLUSION:

Congenital heart defect (CHD) is the most common congenital malformation among all birth defects leading to morbidity and mortality among children. The burden of CHD is high in developing countries like India, due to the high birth rate and critical nature of CHD requiring expensive surgical and non-surgical interventions. Malnutrition and anemia is rampant among children with CHD with a significant impact on the intervention and the outcome of intervention. The high proportion of malnutrition and anemia among children with CHD warrants proper evaluation and early intervention. This is of utmost importance as majority of CHD

are likely to get surgical and non surgical intervention under the RBSK scheme. The RBSK scheme also focuses on malnutrition and deficiency disorder. Accreditation of private institution under the RBSK scheme for intervention of CHD is a big boon to the community

Keywords: Congenital Heart Disease, Nutritionasl Statutus ,Anemia,

LIST OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Congenital heart defect (CHD) is the most common defect among all birth defects representing a major global health problem. Twenty-eight percent of all major congenital anomalies comprise of heart defects[1]. CHD is considered a real challenge because of the complex interplay between medical, surgical, dietetic and socio-economic factors. Most congenital defects are well tolerated in the fetus because of the parallel nature of fetal circulation. Only after birth when fetal pathways begin to close that the full hemodynamic impact of anatomic abnormality becomes apparent.

PREVELANCE:

Congenital heart disease (Cyanotic and Acyanotic) occurs in approximately 0.8% of live births.[2] About 2-3% will be symptomatic by 1st year of life and the diagnosis of CHD is established by 1 week of age in 40-50% and 1 month in 50-60% of patients. Prevalence of CHD in India is reported to be between 2.5 to 5/1000 live births but recent studies by Bhat et al[3] and Smitha et al[4] have suggested the prevalence to be between 8.5 and 13.6.

Table 1: Frequency of Congenital Heart Diseases

Lesion	% of all lesions	Lesion	% of all lesions
Ventricular septal defect	35-30	Hypoplastic left ventricle	1-3
Atrial septal defect(secundum)	6-8	Hypoplastic right ventricle	1-3
Patent ductus arteriosus	6-8	Truncus arteriosus	1-2
Coarctation of aorta	5-7	Total Anomalous pulmonary venous return	1-2
Tetralogy of fallot	5-7	Tricuspid atresia	1-2
Pulmonary valve stenosis	5-7	Single ventricle	1-2
Aortic valve stenosis	4-7	Double outlet right ventricle	1-2
D-Transposition of great arteries	3-5	Others	5-10

MALNUTRITION OF CHD:

Around 59% of children with congenital heart disease were found to be malnourished (weight is affected more than height) irrespective of the cardiac

diagnosis.[41] The risk factors for malnutrition in children with congenital heart disease are multifactorial and comprises of heart failure, cyanosis, multiple heart defects, delayed corrective surgery, anemia and pulmonary hypertension.[6] Children born with congenital heart disease are considered part of a nutritional high-risk group. Elevated energy expenditure caused by the possible causes are: poor socioeconomic status and lack of knowledge of nutritional requirements required for a particular age and sex.[7] Chronic hypoxaemia is an important factor in anorexia and inefficient processing of nutrients.[8] Malabsorption or feeding difficulties.[9] Hyper metabolism probably due to increased catecholamine production and abnormal demands of various organs, in particular the muscles of respiration, the myocardium and hematopoietic system.[10] The severity of malnutrition ranges from mild under nutrition to failure to thrive. There is clear evidence of an association between malnutrition and poor wound healing, impaired immunity, reduced muscle function, and an increased risk of postoperative pneumonia. In the long term, malnutrition in infancy can produce suboptimal growth and physical and cognitive development later in childhood and adolescence.

ANEMIA IN CHD:

Anemia is an important risk factor for morbidity and mortality in patients with cyanotic and acyanotic congenital heart disease. Children with malnutrition commonly have anemia which is attributed to bone marrow hypoplasia, iron, vitamin B12, vitamin A and folate deficiency.[11]Uncorrected congenital cyanotic heart lesions (and some acyanotic lesions with the development of Eisenmenger's complex) keep the body in a state of constant hypoxia which triggers stimulation of bone marrow to produce more RBC's and thus improve oxygen delivery to tissues. RBC indices depend on the cyanosis level.[12] Anemia is an important contributing factor that affects the growth of the child which is frequently missed or underdiagnosed in daily practice.

AIMS AND OBJECTIVES

Objective

- To assess the nutritionals status of children 1-12years with congenital heart disease by anthropometric measurements, and determining Weight for Age(underweight), Height for Age(stunting), Weight for Height(wasting), MUAC and BMI
- To assess degree and type of anemia in study population by assessment of hemoglobin, red cell indices, red cell distribution width and peripheral smear

SCIENTIFIC JUSTIFICATION AND HYPOTHESIS

Congenital heart defect (CHD) is the most common congenital malformation among all birth defects leading to morbidity and mortality among children. The burden of CHD is high in developing countries like India, due to the high birth rate and critical nature of CHD requiring expensive surgical and non-surgical interventions. Malnutrition and anemia is rampant among children with CHD with a significant impact on the intervention and the outcome of intervention

Null Hypothesis: Malnutrition and Anemia is not a common association with CHD

Alternate Hypothesis: Malnutrition and Anemia is commonly associated with CHD

REVIEW OF LITERATURE

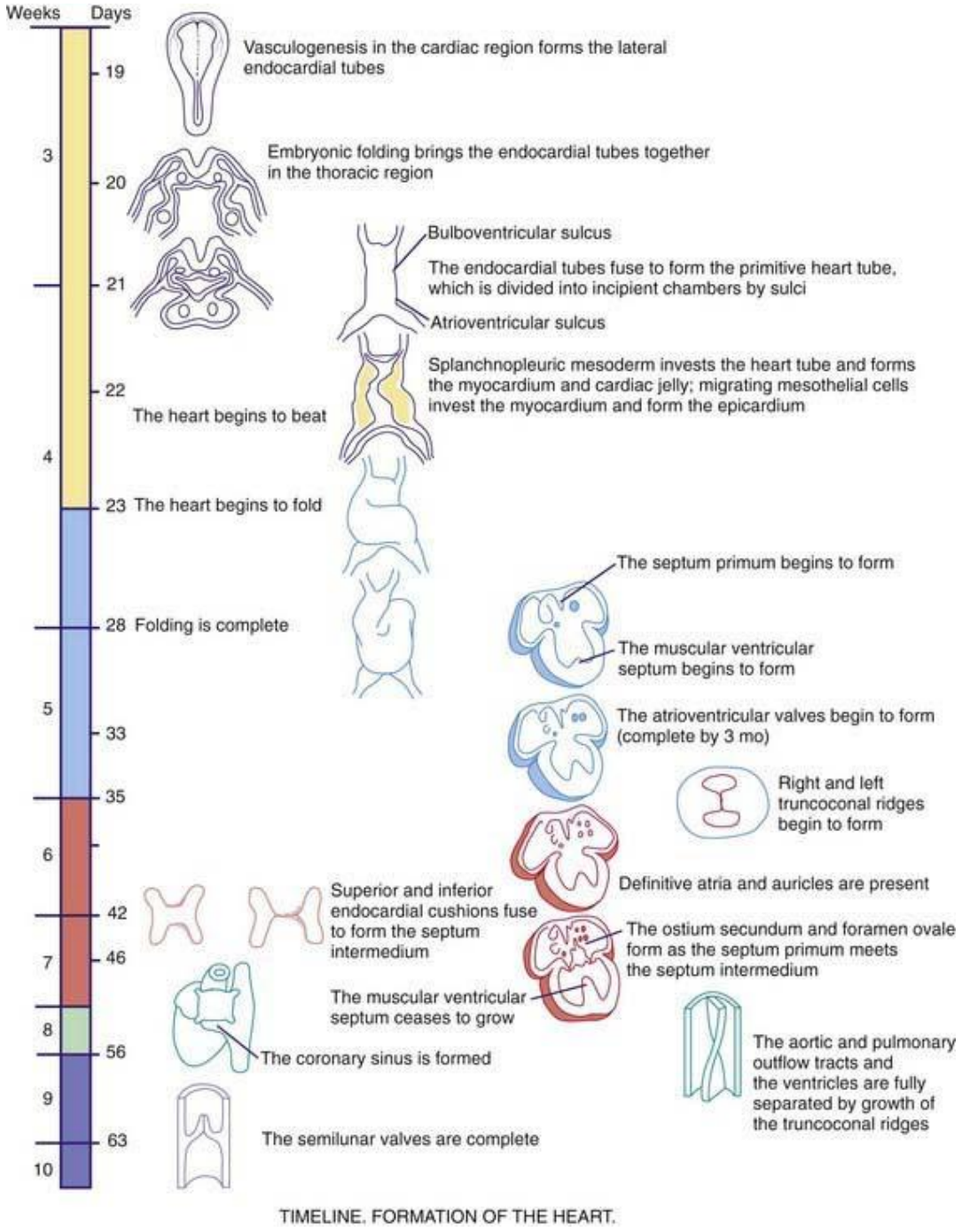
Diseases of the cardiovascular system are an important cause of childhood morbidity and mortality. Majority of heart diseases presenting in early childhood are congenital occurring due to structural defects during development. The management of children with congenital heart diseases require an integrated approach with inputs from various specialities. Many congenital heart defects that are universally considered fatal can be corrected and affected children can expect to survive into adulthood. The developments include improved understanding of the pathophysiology of disease, advances in diagnostic capability, successful surgical and medical management of various heart diseases. Congenital heart disease is considered an important cause of malnutrition that adds to acute and chronic malnutrition along with developmental delay. Early corrective surgery with proper nutrition pre and post-surgery are required for better outcome.

DEVELOPMENT OF HEART:[13]

Knowledge of the cellular and molecular mechanisms of cardiac development is necessary in understanding congenital heart defects and will be even more important in developing strategies for prevention, whether cell or molecular therapies or fetal cardiac interventional procedures

It has these four significant phases:

A. Early cardiac morphogenesis
B. Cardiac looping
C. Cardiac septation
D. Aortic arch development



TIMELINE. FORMATION OF THE HEART.

Fig.1: Timeline formation of heart

A. EARLY CARDIAC MORPHOGENESIS

In the early presomite embryo, cardiac progenitor cell clusters are arranged in anterior lateral plate mesoderm on both side of embryo central axis. By 18 days of gestation, these clusters form cardiac tubes and paired tubes fuse in midline on the ventral surface of the embryo to form the primitive heart tube by 22 weeks of gestation

The embryonic heart begins to contract and exhibit phases of the cardiac cycle by 20-22 weeks of gestation

This straight heart tube is composed of an outer myocardial layer, an inner endocardium, and a middle layer of extracellular matrix known as the cardiac jelly. There are 2 distinct cell lineages:

- Primary heart field which provides precursor cells for the left ventricle
- Secondary heart field provides precursors for the atria and right ventricle.

Premyocardial cells, including epicardial cells and cells derived from the neural crest, continue their migration into the region of the heart tube. Regulation of this early phase of cardiac morphogenesis is controlled in part by the interaction of specific signaling molecules or ligands

B. CARDIAC LOOPING:

At ≈22-24 days, the heart tube begins to bend ventrally and toward the right. Looping brings the future left ventricle leftward and in continuity with the sinus venosus (future left and right atria), whereas the future right ventricle is shifted rightward and in continuity with the truncus arteriosus (future aorta and pulmonary artery).

When looping is complete, the external appearance of the heart is similar to that of a mature heart; internally, the structure resembles a single tube, although it now has several bulges resulting in the appearance of primitive chambers. The common atrium (comprising both the right and left atria) is connected to the primitive ventricle (future left ventricle) via the atrioventricular canal. The primitive ventricle is connected to the bulbus cordis (future right ventricle) via the bulboventricular foramen. The distal portion of the bulbus cordis is connected to the truncus arteriosus via an outlet segment (the conus).The heart tube now consists of several layers of myocardium and a single layer of endocardium separated by cardiac jelly, an acellular extracellular matrix secreted by the myocardium.

ANOMALIES AT THIS STAGE:

- Situs inversus, heterotaxia , usually associated abnormalities in the L-R patterning of the lungs and abdominal viscera.
- Double-outlet right ventricle and double-inlet left ventricle
- Double-outlet left ventricle and double-inlet right ventricle (rare)

C. CARDIAC SEPTATION:

By 26 days, septation of the heart begins with the ingrowth of large tissue masses, the endocardial cushions, at both the atrioventricular and conotruncal junctions. These cushions consist of protrusions of cardiac jelly, Endocardial cells dedifferentiate and migrate into the cardiac jelly in the region of the endocardial cushions, eventually becoming mesenchymal cells that will form part of the atrioventricular valves. The formation of the atrioventricular valve starts when the atria and inlet portion of the ventricle enlarge; the atrioventricular junction (or canal)

lags behind. Complete septation of the atrioventricular canal occurs with fusion of the endocardial cushions.

ANOMALIES AT THIS STAGE:

- Atrioventricular Canal defects(Absence of separation)
- Ebstein Anomaly

SEPATATION OF ATRIA:

Atrial septation starts at approximately 30 days of embryonic life. The common atrium becomes indented externally by the bulbus cordis and truncus arteriosus which will correspond internally with a thin sickle-shaped membrane (SEPTUM PRIMUM) developing in the common atrium on day 35. The septum primum initially has a concave-shaped edge growing toward the atrioventricular canal. This orifice connecting the two atria is called the OSTIUM PRIMUM. Fenestrations appear in the posterosuperior part of the septum forming the OSTIUM SECUNDUM, thus maintaining a communication between the two atria.

The septum primum grows downward toward the endocardial cushion as they start fusing. As the (superior and inferior endocardial cushions) thus dividing the atrioventricular canal into a right and left segments. The ostium secundum and superior vena cava later acquire a more anterosuperior position. These fenestrations then coalesce and form a larger fenestration. Meanwhile, another sickle-shaped membrane develops on the anterosuperior wall of the right atrium, just right of the septum primum and left of the sinus venosus valve. It grows and covers the ostium secundum, which continues to allow blood passage since the two membranes do not fuse. The septum secundum grows toward the endocardial cushion, leaving only an

area at the posterosuperior part of the interatrial septum where the septum primum continues to exist as the foramen ovale membrane, through which fetal blood passes from the inferior vena cava to the left atrium The septum primum disappears from the posterosuperior portion of interatrial septation and the edge of the septum secundum forms the rim of the fossa ovalis on approximately day 42 of development.[14]

ANOMALIES AT THIS STAGE:

- Atrial Septal Defect

SEPTATION OF VETRICLES:[13]

Septation of the ventricles begins at about embryonic day 25 with protrusions of endocardium in both the inlet (primitive ventricle) and outlet (bulbus cordis) segments of the heart. The inlet protrusions fuse into the bulboventricular septum and extend posteriorly toward the inferior endocardial cushion, where they give rise to the inlet and trabecular portions of the interventricular septum.

ANOMALIES AT THIS STAGE:

- Ventricular Septal Defect

The outlet or conotruncal septum develops from ridges of cardiac jelly, similar to the atrioventricular cushions. These ridges fuse to form a spiral septum that brings the future pulmonary artery into communication with the anterior and rightward right ventricle and the future aorta into communication with the posterior and leftward left ventricle. Differences in cell growth of the outlet septum lead to lengthening of the segment of smooth muscle beneath the pulmonary valve (conus), a process that separates the tricuspid and pulmonary valves. In contrast,

disappearance of the segment beneath the aortic valve leads to fibrous continuity of the mitral and aortic valves

ANOMALIES AT THIS STAGE:

- Truncus arteriosus
- Tetralogy of Fallot
- Pulmonary atresia
- Double-outlet right ventricle
- Interrupted aortic arch
- Group of cardiac anomalies often associated with deletions of the DiGeorge critical region of chromosome 22q11.

D. AORTIC ARCH DEVELOPMENT:[13]

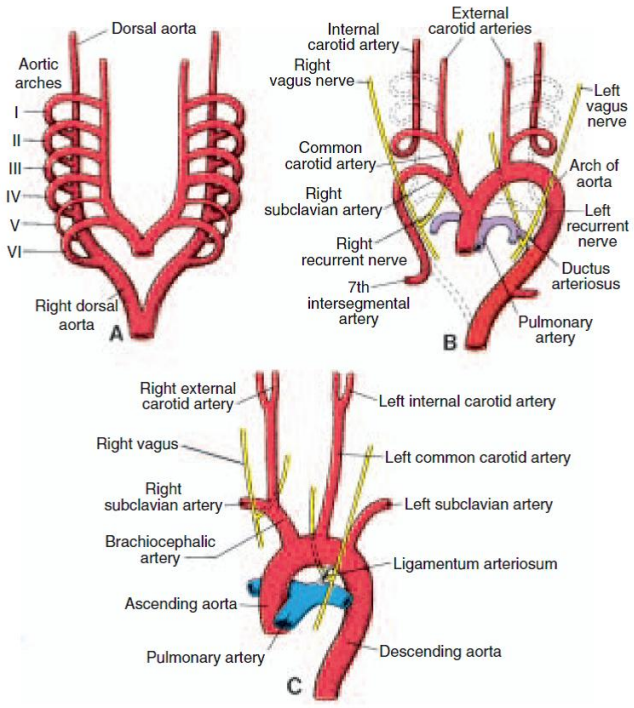


Fig.2.Aortic Arch development A)6 weeks B)7 weeks C) 6month old infant

The aortic arch, head and neck vessels, proximal pulmonary arteries, and ductus arteriosus develop from the aortic sac, arterial arches, and dorsal aortae. When the straight heart tube develops, the distal outflow portion bifurcates into the right and left 1st aortic arches, which join the paired dorsal aortae .The dorsal aortae will fuse to form the descending aorta. The proximal aorta from the aortic valve to the left carotid artery arises from the aortic sac. The 1st and 2nd arches largely regress by about 22 days

- 1st aortic arch giving rise to the maxillary artery
- 2nd aortic arch to the stapedial and hyoid arteries.
- 3rd arches participate in the formation of the innominate artery and the common and internal carotid arteries.
- The right 4th arch gives rise to the innominate and right subclavian arteries, and the left 4th arch participates in formation of the segment of the aortic arch between the left carotid artery and the ductus arteriosus.
- The 5th arch does not persist as a major structure in the mature circulation.
- The 6th arches join the more distal pulmonary arteries, with the right 6th arch giving rise to a portion of the proximal right pulmonary artery and the left 6th arch giving rise to the ductus arteriosus.
- The aortic arch between the ductus arteriosus and the left subclavian artery is derived from the left-sided dorsal aorta, whereas the aortic arch distal to the left subclavian artery is derived from the fused right and left dorsal aortae.

ANOMALIES AT THIS STAGE:

- Hypoplastic Ascending Aorta
- Coarctation of the Aorta

- Interrupted Aortic Arch
- Patent Ductus Arteriosus
- Double aortic arch

FETAL CIRCULATION: [15]

The fetal circulation and the postnatal changes occurring thereafter is significant in our understanding of the various congenital heart diseases. Most of these information has been obtained by studies on the foetal heart due to the similarity it bears with human fetal circulation. The heart begins to function by the end of the sixth week and the fetal circulation is established. The unique features of the fetal circulation are

- ❖ Presence of placental circulation
- ❖ Absence of gas exchange in the lungs
- ❖ Presence of ductus venosus
- ❖ Widely open foramen ovale
- ❖ Widely open ductus arteriosus

ANATOMY AND PHYSIOLOGY OF THE FETAL CIRCULATION:

The fetal blood is carried to the placenta by two umbilical arteries and returned from the placenta by the persistent left umbilical vein. The umbilical vein enters the abdomen at the umbilicus and passes along the free margin of the falciform ligament into the liver. At the porta hepatis, it is joined by the left branch of portal vein and from opposite this point the ductus venosus arises and joined by the left hepatic vein before it opens into the inferior vena cava

In the inferior vena cava, there is mixing of blood brought by the ductus venosus and hepatic veins with the blood returning from the lower limbs and abdominal wall. It enters the right atrium, and guided by the valve of the inferior vena cava, passes mostly through the foramen ovale into the left atrium, where it mingles with small amount of blood returned from the lungs by the pulmonary veins. Some amount of blood from inferior vena cava along with blood from superior vena cava goes through the right atrioventricular orifice to the right ventricle.

The blood in the left atrium passes into the left ventricle and from there into the aorta and this is distributed to heart itself, the head and the upper limbs. The blood from here are returned to the right atrium by the superior vena cava. The blood in the right ventricle is conveyed into the pulmonary trunk. The fetal lungs being inactive only a small amount of blood goes into the pulmonary vasculature and is returned to the left heart by pulmonary veins.

The greater part of the blood from the pulmonary trunk passes through the ductus arteriosus into the aorta where it mixes with the small quantity of blood transmitted by the left ventricle into this part of the aorta. It descends through the aorta and is in part distributed to the lower limbs and to the viscera of the abdomen and pelvis, but most of it is conveyed by the umbilical arteries to the placenta.

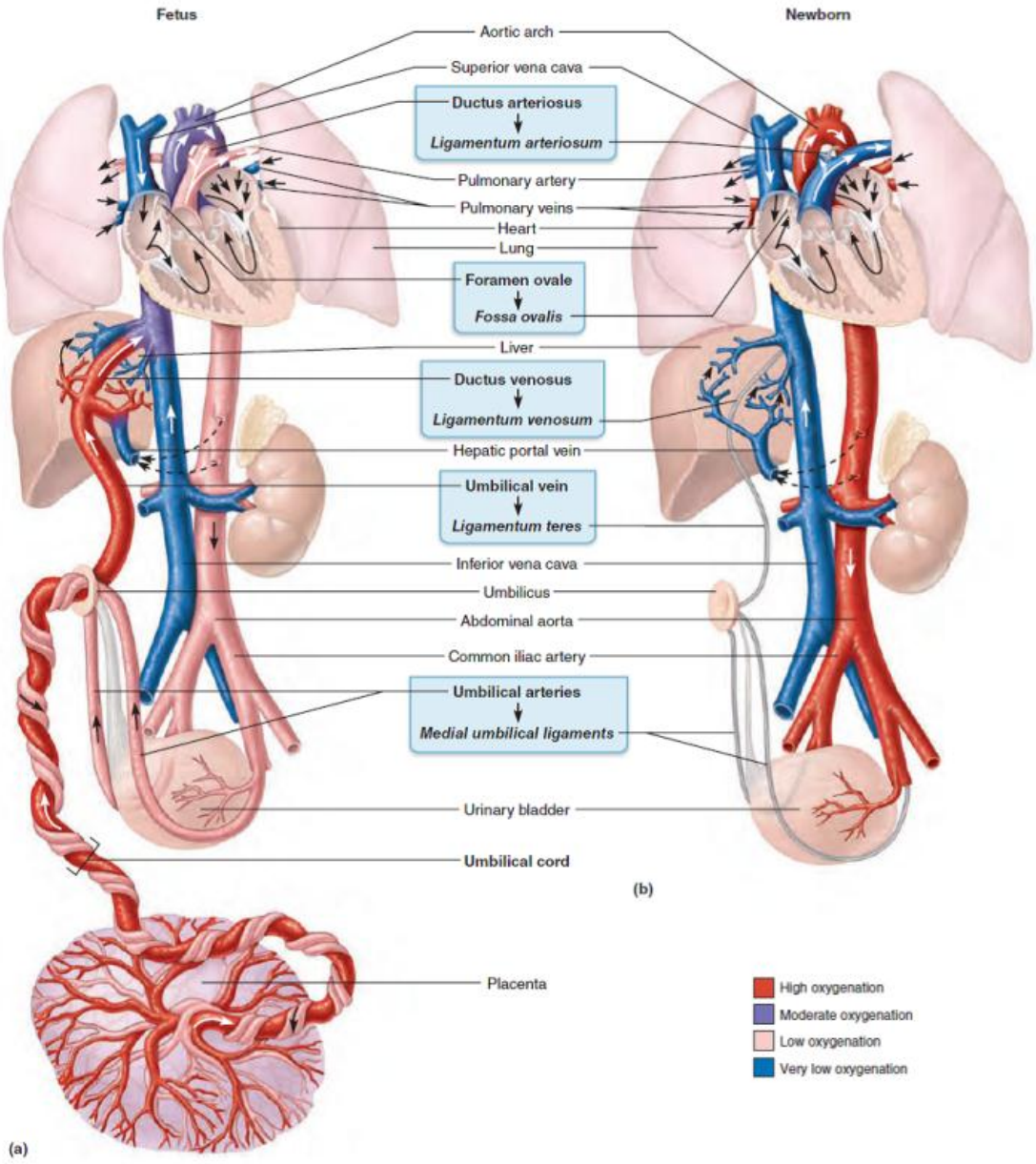


Fig. 3: Fetal Circulation

DISTRIBUTION OF BLOOD

The inferior vena caval blood represents 70% of the total venous return to the heart. The superior vena caval blood about 22-25% of the total venous return. 1/3rd of the inferior vena caval blood passes through the foramen ovale to combine with

8% of the venous return from the pulmonary veins and then passes into the left ventricle and enter the aorta. The remainder of the inferior vena caval blood and superior vena caval blood enter the pulmonary trunk after passing into the right ventricle. The left ventricular output is approximately half of the right ventricle. The left ventricular output is approximately half of the right ventricular output or one third of the combined left and right ventricular output.

OXYGEN SATURATION AND PARTIAL PRESSURE OF OXYGEN:

The blood in the umbilical vein has an oxygen saturation of 80% PO_2 32 -35. After combining with the blood coming from the lower extremities, the oxygen saturation drops to 70% within the inferior vena cava. The blood from the superior vena cava has an oxygen saturation of 40% and PO_2 of 12 to 14. The blood that enters the pulmonary trunk contains 50-55% saturation and PO_2 of 16 to 18. The saturation of blood in the descending aorta is about 55 to 60% and has a PO_2 of 20-22 %.

INTRAVASCULAR PRESSURES IN THE FETUS

In the fetus, the pressure in the aorta and pulmonary trunk are identical (70/45mmHg) because they are connected by a wide ductus arteriosus. The right and left ventricles also have an identical systolic pressure of 70mmHg. The right atrial mean pressure (4mmHg) is slightly higher than the left atrial mean pressure (3mmHg) and this aids in flow of blood through the foramen ovale from right to left. The fetal measurements are in relations to the intra- amniotic pressure whereas, postnatally they are related to the atmospheric pressure.

THE CHANGES IN THE VASCULAR SYSTEM AT BIRTH

At birth, when respiration is established and increased amount of blood from the pulmonary trunk passes through the pulmonary arteries to the lungs and correspondingly increased amount returns by the pulmonary veins to the left atrium. At the same time there is a fall in pressure in the inferior vena cava due to reduction of the venous return by occlusion of the umbilical veins, the pressure within the two atria become equalised and the foramen ovale, which is valve like in character is closed by apposition, and later by the fusion, of the septum primum to the septum secundum.

When the umbilical cord is ligatured and the placental circulation is cut off, the umbilical veins become thrombosed and is gradually converted into a fibrous cord which constitutes the ligamentum teres of the liver. The umbilical vessels constrict in response to a variety of stimuli, such as handling, stretching, cooking and altered oxygen and carbon dioxide tensions in the blood. The ductus venosus also become obliterated but the mechanism is not known. Its remnant is seen as the ligamentum veosum of the liver in adults.

The umbilical arteries on ligature of the umbilical cord are thrombosed from the point at which they give their last branches the superior vesical arteries to the umbilicus. They are subsequently converted into fibrous strands and produce the medial umbilical folds of peritoneum. Obliteration of the ductus arteriosus is also essential. This channel contracts rapidly at first. The direction of blood flow in this vessel is reversed, due to a rise in the systemic vascular resistance, resulting from exclusion of the placental circulation and a fall in the pulmonary resistance with expansion of the lungs. Anatomical closure of the ductus arteriosus occurs by

proliferation of the lining endothelium but takes some months to complete. The initial constriction of the ductus arteriosus at birth has been attributed to a direct effect of the raised oxygen tension in the blood on its muscular wall. Ultimately it forms an impervious cord which connects the left pulmonary artery with the arch of the aorta and is termed as the ligamentum arteriosum.

RISK FACTORS IN CHD:

The cause of most congenital heart defects remains unknown. They are multifactorial and result from a combination of genetic predisposition and yet to be determined environmental stimulus. A small percentage of congenital heart lesions are associated with certain chromosomal abnormalities, in particular, trisomy 21(50%),13 and 18(90%) and turner syndrome(40%).[2]

Of all cases of congenital heart diseases, 2-4% are associated with known environmental or adverse maternal conditions and teratogenic influences including maternal diabetes mellitus, phenylketonuria, systemic lupus erythematous, congenital rubella syndrome and maternal ingestion of drugs (lithium, ethanol, warfarin, thalidomide, anti convulsant agents.[2]

Gnanalingam MG et al, , Department of Paediatrics, Booth Hall Children's Hospital, Manchester UK,1997,executed a case control study to assess the association between congenital heart disease and parental consanguinity in South India and observed that parental consanguinity was noted in 12.5% of the control group compared to 31.1% of the CHD group.[16]

Susan M. Becker et al ,Epidemiology Section, King Faisal Specialist Hospital and Research Center, Saudi Arabia,2001, did a cross sectional study that

examined a large number of congenital heart disease patients in an inbred population to investigate the relationship between First-cousin Marriage and subgroups of CHD. They observed that out of the subgroups of CHD , first-cousin consanguinity was significantly associated with ventricular septal defect (VSD), atrial septal defect (ASD), atrioventricular septal defect (AVSD), pulmonary stenosis (PS), and pulmonary atresia (PA) and concluded that in a population with a high degree of inbreeding, consanguinity may exacerbate underlying genetic risk factors, particularly in the offspring of first cousins and there may be a recessive component in the causation of some cardiac defects.[17]

Khalid Y. et al, Department of Paediatrics, Faculty of Medicine, American University of Beirut Medical Center, Lebanon, 2002, did a case control study to assess the independent effect of consanguineous marriage on the overall prevalence of CHD as well as specific subtypes of CHD. They observed that infants born to first cousin marriages had a 1.8 times higher risk of having a CHD diagnosed at birth compared to those born to unrelated parents.[18]

Chehab G et al, Department of Paediatrics, Lebanese University, Faculty of Medical Sciences, 2007, conducted a case control cohort study to determine the incidence of congenital heart disease and inbreeding in Lebanon and found that subgroups with first degree cousins, first plus second degree cousins, and any degree of consanguinity, are significantly larger in the cohort with congenitally malformed hearts than in the control cohort.[19]

The risk of occurrence increases if a 1st degree relative (parent/sibling) is affected (2-6%).When two first degree relatives are affected with CHD, the risk of subsequent child may reach to 20-30%.[2]

Oyen N.et al, Department of Epidemiology Research Copenhagen, Denmark, 2005, carried out a national cohort study to investigate whether an individual's risk of being born with specific heart defects is influenced by prior heart defects in family members and observed that specific CHDs (ASD, VSD, Hetrotaxia, conotruncal defects) showed highly variable but strong familial clustering in first-degree relatives, ranging from 3-fold to 80-fold compared with the population prevalence.[20]

Peyvandi S, Division of Pediatric Cardiology, Department of Pediatrics, The Children's Hospital of Philadelphia, Pennsylvania,2010,carried out a cross sectional study to estimate the risk of CHD in relatives of patients with conotruncal defects in the current era, and to assess whether specific subtypes of conotruncal defects influence risk in first-degree relatives. They observed that the risk of CHD was higher in siblings than in parents and also recurrence risk varies by lesion and relationship.[21]

Ellesøe GS et al, Programme for Disease Systems Biology, University of Copenhagen, Denmark, 2016, executed a observational study to determine if familial co-occurrence of lesions follows specific patterns. They observed that distinct groups of cardiac malformations co-occur in families, suggesting influence from underlying developmental mechanisms.[22]

Miller A et al**,** Division of Birth Defects and Developmental Disabilities, Center for Disease Control and Prevention, Atlanta, 2005, USA, carried out a cohort study to examine the effect of increasing maternal age on the prevalence for several isolated (nonsyndromic) CHD phenotypes, and explore potential effect modification of the maternal age group-specific CHD prevalence estimates by preterm birth,

LBW, maternal race, and infant sex. They observed that birth prevalence of isolated CHDs in the aggregate might be associated with advanced maternal age, especially 35 years of age or older and also infants born to mothers older than 35 years of age seemed to be at 20% increased risk of CHDs, while infants born to younger mothers tended to be at decreased risk of CHDs, along with increased prevalence of specific groups of CHDs, such as laterality defects, conotruncal defects, LVOTOs, RVOTOs, AVSDs, and VSDs.[23]

Gil KS et al, National Center on Birth Defects and Developmental Disabilities, Centers for Disease Control and Prevention, Atlanta, USA, 2007, did a population based study to assess the association between maternal age and the risk for birth defects of unknown etiology and observed that older maternal age, ≥40 years, is associated with some atrial and ventricular septal defects, Tetralogy of Fallot, oesophageal atresia, craniosynostosis, and hypospadias.[24]

Luo YL et al, Department of Epidemiology, Southern Medical University, Guangzhou, China, 2012, carried out a case control study to investigate the relationship between maternal age, parity and selected isolated birth defects through a large cohort from Baoan district, Shenzhen, China and concluded that a protective effect on the occurrence of CHD was seen in younger mothers.[25]

Abqari S et al, 2015, did a case control to study the profile of patients with CHD in western Uttar Pradesh and also the risk factors associated with it so that preventive measures can be taken to prevent CHD during pregnancy. They found a significant association between incidence of CHD and advanced parental age, bad obstetric history, febrile illness during pregnancy, and a folic acid‐deficient diet.[26]

Best K.E et al, Institute of Health & Society, Newcastle University, United Kingdom, 2016, did a population based cross sectional study, to examine the association between maternal age at delivery and CHD prevalence in the North of England. They concluded that advanced maternal age is not a risk factor for CHD, however, there was a marginal increase in the prevalence of common arterial trunk, PDA, pulmonary valve stenosis; and single ventricle among mothers aged more than 35years .[27]

Steurer AM et al, Departments of Paediatrics, University of California, USA, 2012, carried out a cohort study to quantify Gestational age specific mortality and neonatal morbidity in infants with cyanotic congenital heart disease. They concluded that incidence of CCHD was highest at 29 to 31 weeks' GA (0.9%) and lowest at 39 to 42 weeks (0.2%) and that morbidity remains increased across all Gestational groups in comparison with infants born at 39 to 42 weeks.[28]

Auger N et al, Department of Social and Preventive Medicine, University of Montreal, Canada, 2012, did a case control s study to determine the relationship between preeclampsia and prevalence of congenital heart defects in offspring. They found that an elevated prevalence of heart defects(affecting all general structures of the heart, including the aorta, pulmonary artery, valves, ventricles, and septa) among infants of women with preeclampsia compared to those with no preeclampsia . Women with early-onset preeclampsia had significantly greater prevalence of infants with heart defects, both critical and noncritical, compared with those with no preeclampsia, whereas women with late onset had only marginally greater prevalence.[29]

Ramakrishnan A et al, Division of Epidemiology, University of Texas School of Public Health, TX, USA,2013, conducted a systematic review and meta-analysis to assess the associations between untreated and treated maternal hypertension and the risk of CHDs, evaluating CHDs overall as well as specific CHD subtypes and came to an conclusion that significant associations were observed between maternal hypertension and overall CHDs, for both treated and untreated hypertension, as well as for overall hypertension regardless of treatment status.[30]

Boyd et al, Department of Epidemiology Research, Statens Serum Institute, Denmark, 2017, conducted a cohort study to identify specific offspring heart defect subtypes associated with maternal hypertensive disorders of pregnancy and to pinpoint whether maternal or fetal mechanisms contribute most to the observed associations. They found that offspring congenital heart defects were associated with a 7-fold increase in the risk of early preterm preeclampsia in the same pregnancy, an almost 3-fold increase in late preterm preeclampsia risk, and a 16% increase in term preeclampsia risk compared with carrying a child with no heart defect.[31]

Tabib A et al, Heart Valve Disease Research Center, Rajaie Cardiovascular Medical and Research Center, Iran, 2012, carried out a case control study to determine the prevalence of cardiac malformations in fetuses of Iranian diabetic mothers with pre-gestational and gestational diabetes mellitus (GDM) and to find the patterns of different cardiac malformations. They detected fetal cardiac malformations in 8.8% of mothers with diabetes mellitus.[32]

Muhammed A et al, Department of Paediatrics, Lady Reading Hospital, Pakistan, 2013, did a cross sectional study to determine the frequency of congenital heart disease in infants of diabetic mothers referred to Pediatrics department. They

observed that frequency of congenital heart disease in IDMs was 52.5% out of which PDA, VSD and ASD were more common.[33]

Hunter EL et al, Specialist Registrar in Paediatric and Fetal Cardiology, Evelina London Children's Hospital, UK, 2015, did a retroprespective observational study to compare the occurrence of congenital heart disease in the two referral groups and to evaluate whether detailed fetal echocardiography should be offered to women with GDM and observed that risk of CHD in a GDM pregnancy is 2.76% with a 26.7% risk of a concomitant extra cardiac abnormality.[34]

Arjmandnia M et al**,** Department of Paediatrics, Iran,2 018,conducted a case control study to investigate the determinant factors leading to CHD and concluded that a significant association was present in mothers with a Family History of CHD, from consanguineous marriages, history of diabetes, antiepileptic use, and lack of folic acid use. However, no significant associations were found between new-born CHD and maternal age, BMI, or cigarette smoking.[35]

PATHOPHYSIOLOGY OF MALNUTRITION IN CHD:

Forchielli, M. L.et al, Paediatric Gastroenterology and Nutrition Hospital and Children's Hospital, USA,1994,explained in her article the mechanism of malnutrition in CHD depends on type of cardiac lesion (cyanotic versus acyanotic); Low energy intake formations due to loss of appetite ,anoxia and peripheral acidosis , mal absorption, relative increased nutrient requirements; hyper metabolism due to multiple infective episodes ,increased oxygen consumption, increased basal body temperature, low body fat stores ,age at time of operation, prenatal factors such as parental height, genetic factors, intrauterine factors, and birth weight.[36]

Jackson M et al, 2007, Liverpool, UK, conducted a study on failure to thrive in infants with congenital heart disease attributed by their low energy intakes and high resting expenditures. They found that mean gross energy intakes increased by 31.7% on high energy feeding and mean weight gain improved from 1.3g/kg/day to 5.8g/kg/day.[37]

Hubschman L, Columbia University School of Nursing, New York, 2013, explained in her article that the pathophysiology of malnutrition in CHD was mainly due to increased metabolic demand which was not matched by the caloric and protein intake, inadequate nutrient intake due to delayed feeding milestones & gastrointestinal morbidity, volume overload on the right ventricle which decrease cardiac output and amplify the risk for decreased splanchnic perfusion leading to insufficient nutrient absorption and also comorbidities such as malnutrition and growth failure have the potential to significantly affect morbidity and mortality secondary to CHD.[38]

PREVELANCE OF MALNUTRITION:

Mitchell IM et al, Yorkhill, Glasgow, 1995, did a cross sectional study to assess the nutritional status of children with congenital heart disease. They concluded that a marked degree of under nutrition was evident in all children; 52% had weight less than the third centile, 37% were below the third centile for height, and 12-5% were below the third centile for triceps skin fold thickness and 18-8% for subscapular skin fold thickness. Mid arm circumference and arm muscle circumference were below the fifth centile in 20.1% and 16.7% of children respectively. Five or more of the 29 biochemical and hematological measurements were abnormal in 83-3% of patients; 10 or more were abnormal in 12-5% of patients.[39]

Varan B et al, Department of Pediatrics, Baskent University School of Medicine, Turkey, 1998, investigated the effect of several types of congenital heart disease (CHD) on nutrition and growth .They found that Mild or borderline malnutrition was more common in group acyanotic CHD with pulmonary hypertension patients. Both moderate to severe malnutrition and failure to thrive were more common in group cyanotic with pulmonary hypertension patients.[9]

Baaker RH et al, Department of Pediatrics Al Mustansirya Medical College, Iraq, 2002,executed a descriptive study to assess the growth (Weight, height and head circumference) in patients with different types of congenital heart disease (CHD).They observed acute malnutrition(29.5%) is more obvious than chronic malnutrition(21.9%) in patients with CHD, acute malnutrition is more common in patients with a cyanotic CHD without HF or PH (39.2%), while chronic malnutrition is more obvious in patients with PH and HF (26.3%) and (25%) respectively.[40]

Vaidyanathan B et al, Amrita Institute of Medical Sciences and Research Center, Kerala, India, 2007, did a prospective study to identify determinants of malnutrition in children with congenital heart disease (CHD) and examine the short-term effects of corrective intervention. They concluded irrespective of the cardiac diagnosis, malnutrition is common in children with CHD (59%). [41]

Okoromah CA et al, Department of Pediatrics, University of Lagos, Nigeria, 2008, carried out a case control study to investigate the prevalence, profile and predictors of severe malnutrition in children with congenital heart defects (CHDs).They observed 90.4% of cases and 21.1% of controls had malnutrition , and 61.2% and 2.6%, respectively, had severe malnutrition. Wasting was significantly higher (58.3%) in acyanotic CHD), and stunting (68.0%) in cyanotic CHD. [42]

Vaidyanathan B et al, Amrita Institute of Medical Sciences and Research Center, Kerala, India, 2009, did another cross sectional study to examine the impact of corrective intervention on the nutritional status of children with CHD and identify factors associated with suboptimal recovery. They deduced that severe malnutrition was present in over half of the patients with CHD and is not always reversed by corrective surgery or intervention. Persistent malnutrition after corrective intervention is determined by nutritional status at presentation, birth weight, and parental anthropometry.[43]

Ratanachu-ek S et al, Department of Paediatrics, Thailand, 2011, conducted a prospective cohort study to evaluate the impact of cardiac surgery on nutritional status of paediatric patients with CHD. They concluded that malnutrition was present in malnutrition 40% of the children with CHD (underweight 28%, wasting 22% and stunting 16%) and cardiac surgery has a significant positive effect on weight gain and nutritional status.[44]

Daymont C et al, Department of Paediatrics and Child Health, University of Manitoba, Canada, 2012, performed a retrospective matched cohort study, identifying children with CHD in a large primary care network in Pennsylvania, New Jersey, and Delaware and matching them 10:1 with control subjects. They concluded that children with CHD experience early, simultaneous decreases in growth trajectory across weight, length, and head circumference. The simultaneous decrease suggests a role for altered growth regulation in children with CHD.[45]

Ijeoma Arodiwe et al, University of Nigeria teaching hospital, Nigeria, 2014, executed a case control study to determine the burden and determinant of malnutrition in children with several types of congenital heart disease (CHD).They

observed that prevalence of malnutrition was 92% in children with CHD out of which 60% contributed to severe malnutrition and also discovered that the potent trigger of severe malnutrition among children with congenital heart disease (though more in cyanotic heart disease) is pulmonary hypertension.[46]

Batte et al, Child Health and Development Center, Makerere University College of Health Sciences, Uganda, 2014, carried out a cross sectional study to determine the prevalence of malnutrition in children with congenital heart disease and the factors associated with wasting, underweight and stunting and found that 42.5% children were underweight, 45.4% children were stunted, 31.5% were wasted and 27.1% were thin (according to BMI).[47]

Hassan B.A. et al, Zagazig University, Zagazig, Egypt, 2015, did a case–control study to identify prevalence and predictors of malnutrition in Egyptian children with symptomatic CHD. They concluded that prevalence of malnutrition was 84.0% in patients with CHD and 20% in controls. In patients with acyanotic CHD, stunting was proportionately higher (57.89%) than in cyanotic CHD, while wasting was predominant (45.83%) in the latter.[48]

Oyarzún I. et al, Department of Pediatrics, Faculty of Medicine, Pontificia University, Chile, April 2016, carried out a longitudinal study of a concurrent cohort to describe nutritional recovery in children with CHD and associated factors after surgery and found that there was a significant percentage who were underweight(28.3%) and had short stature (21.7%) at the time of admission indicating both acute and chronic malnutrition and the nutritional recovery post-surgery had an effect on weight for age and not on the length/height of the children included in the study.[49]

Swagata M et al, Department of Paediatrics, Vydehi Institute Medical Sciences and Research Center, Karnataka, India, July 2016, carried out a case-control observational study to assess the effect of CHD on growth and nutrition and to identify the areas of growth affected with reference to different anthropometric measurements. After data analysis, arrived at a conclusion that a significantly higher, 82% were underweight and 86% were stunted among children with congenital heart disease with a statistical significance of ($P<0.001$).[50]

Habeeb NM et al, Paediatric Cardiology, Ain Shams University, Egypt. Nov 2017, conducted a cross sectional study to evaluate the nutritional status of CHD patients. They observed that malnutrition, stunting and wasting were detected in 65.8%, 66.4% and 62.5% of patients respectively and prevalence rates were significantly higher among cyanotics (62.8%, 74.4% and 25.6%) when compared to acyanotics (49.5%, 63.3% and 18.3%) respectively with a p value < 0.05.[51]

Noori M et al, Children and Adolescent Health Research Center, Zahedan University of Medical Sciences, Iran, Dec 2017, executed a case control study to compare growth status between children with CHD and healthy children. They interpreted that weight and head circumference were significantly lower in CHD children compared to healthy children ($p<0.05$).[52]

Begum R et al, Department of Paediatrics, Jawaharlal Nehru Medical College, Dec 2017, Maharashtra, India, did a case control study to assess the nutritional status of the children with CHD and observed stunting was evident in 58.72% cases and in 41.26% of controls whereas 82.53% of cases and 24.6% of controls were underweight.[53]

PREDICTORS OF MALNUTRITION IN CHD

Heart failure is the common predicator of malnutrition due to increased metabolic needs of these children but low energy intake due to their inability to tolerate a high volume of feeds. The body in a state of constant hypoxia which triggers stimulation of bone marrow to produce more RBC's and thus improve oxygen delivery to tissues and with time iron stores are depleted leading to microcytic anemia which further aggravates malnutrition in these children.

Bukovac LT et al, Department of Paediatrics, Croatia, 1993, conducted a cross sectional study to determine the predictors of malnutrition in children with CHD and found that the type of congenital heart disease complicated with pulmonary hypertension had a significant correlation with malnutrition.[54]

Leite HP et al, Department Of Paediatric Cardiology, Brazil, 1995, conducted a prospective study to assess the nutritional status of children with congenital heart disease with left-to-right shunt and the nutritional disturbances related to the presence of pulmonary hypertension (PH).They observed that overall prevalence of malnutrition of 83.3%, which was higher in those with PH and that the presence of PH was associated with higher nutritional disturbance.[55]

Vaidyanathan B et al, Amrita Institute of Medical Sciences and Research Center, Kerala, India, 2007, observed that predictors of malnutrition at presentation are congestive heart failure (CHF), age at correction, lower birth weight and fat intake, previous hospitalizations, small for gestation, lower maternal height and fat intake, genetic syndrome.[41]

Okoromah CA et al, Department of Paediatrics, University of Lagos, Nigeria, 2008, stated that the predictors of malnutrition in CHD were anaemia,

moderate to severe congestive heart failure (CHF), poor dietary intake and prolonged unoperated disease.[42]

Batte et al, Child Health and Development Center, Makerere University College of Health Sciences, Uganda,2014, carried out a cross sectional study to determine the prevalence of malnutrition in children with congenital heart disease and the factors associated with wasting, underweight and stunting and found that heart failure and anemia were the most common predicators of malnutrition.[47]

Hassan BA et al, Egypt, 2015, did a case–control study to identify prevalence and predictors of malnutrition in Egyptian children with symptomatic CHD and concluded that malnutrition correlated significantly with low haemoglobin level, low arterial oxygen saturation, heart failure, pulmonary hypertension, and poor dietary history.[48]

Blasquez A et al, April 2016, did a cohort study to identify the prevalence of malnutrition in children with congenital heart disease and observed that moderate to severe malnutrition is present in 15% of children with CHD, and it is more frequent in case of pulmonary hypertension. Half of these children demonstrate low caloric intake, whereas few have proper nutritional support .[56]

Begum R et al, Department of Pediatrics, Jawaharlal Nehru Medical College, Dec 2017, Maharashtra, India, also observed that malnutrition correlated significantly with heart failure, low haemoglobin level, poor dietary history and pulmonary hypertension and was statistically highly significant.[53]

El-Koofy N et al, Department of Pediatrics, Cairo University, Egypt, 2017, carried out a cohort study to assess the nutritional status of group of infants with

acyanotic CHD associated with increased left to right shunt and evaluate the impact of nutritional counselling for those infants on their nutritional status. They observed that congestive cardiac failure, repeated chest infections and delayed surgery were main predictors of malnutrition and overall prevalence of malnutrition (according to Waterlow criteria for failure to thrive) was found to be present in 62% of the studied population.[57]

Balogun MF et al, Department of Pediatrics, University College Hospital, Nigeria, 2018, did a case control study, to assess nutritional status of children with CHD and apparently healthy controls, compare nutritional indices of these groups of children as well as parental ages and heights. They deduced that low socioeconomic status, type of congenital heart especially cyanotic congenital heart disease and delayed surgical intervention correlated with incidence of malnutrition in children with CHD.[58]

ANEMIA AND CHD:

Gaiha et al, 1993, did a clinico-hematological study of iron deficiency anemia and its correlation with hyper viscosity symptoms in cyanotic congenital heart disease, reported a prevalence of 18.2% anemia in children with congenital Heart disease.[59]

M.O. Lano et al, Nairobi, East Africa, 2009, found that there is high prevalence of 16.9% of iron deficiency in children with cyanotic heart disease.[60]

H Amoozgar et al, Shiraz University of Medical Sciences, Shiraz, IR Iran, 2011, did a retrospective study to determine the prevalence of unrecognized anemia in paediatric patients with different congenital heart diseases who referred for cardiac surgery. They noticed that a significant number of patients with CHD had

anemia preoperatively without being considered by physicians. Many investigators found low dose iron therapy or preoperative administration of a single dose of recombinant human erythropoietin without autologous blood donations was beneficial by increasing haematocrit level.[61]

Binh TQ et al, Cardiovascular Center, University Medical Center, Vietnam, March, 2017, conducted a cross sectional study to investigate the prevalence of Iron Deficiency Anemia (IDA) in children with CHD and to assess the diagnostic values of hemogram, especially the erythrocyte indexes as a simple tool for early recognition of IDA. They established that up to 36.1% children in cyanotic group and 24.2% in acyanotic group were diagnosed with true IDA or showed depletion of body iron storage and 77.8% children in cyanotic group and 87.8% in acyanotic group were at risk of iron deficiency and concluded that both groups of cyanotic and acyanotic CHD are equally at high risk of IDA and depletion of body iron storage.[62]

MATERIAL AND METHODS

Study Design: A cross sectional study-purposive sampling and consecutive cases

Study Period: 18 months

Study Group: One

Inclusion criteria:

- Preoperative cases of congenital heart disease within the age group 1-12 years who consult as Outpatients as well as Inpatient for surgical and non-surgical interventions in Pediatrics Department, SMIMS.

b. Exclusion criteria:

- Patients with chromosomal/genetic disorders/congenital syndromes
- Post-operative cases of congenital heart disease and trivial heart diseases
- Patients with acquired heart diseases
- Patients not given consent

Sample Size Calculation:

Sample size (n) = $4pq/d^2$,

Where, p = prevalence (of malnutrition)

q = 100 – prevalence

d = precision is 11

Substituting in the formula, (n) = $4pq/d^2$

P =59%[41], Q=41

The sample size was calculated to be: 79.9 ≈ 80

Study Procedure:

Data was collected at time of consultation of children with congenital heart disease who fulfilled the inclusion criteria in Pediatric OPD as well as admission in Sree Mookambika Institute of Medical Sciences by direct interview of parent of the child using structured questionnaire along with clinical examination of the child, which includes weight which was measured using electronic weighing scale, Height which was measured using infantometer/stadiometer,

MUAC was measured by using non-stretchable, flexible tape. IAP (5-18 years) & WHO (0-5yrs) Growth Charts for Weight for Age, Height for Age, Weight for Height and BMI was utilized for plotting the measurements, Anemia (type and degree) assessed by hematological parameters (CBC, RBC Indices, RDW and Peripheral smear) from the lab investigations which was sent as a part of the clinical evaluation.

The study was initiated after obtaining ethical clearance from the institutions ethical clearance committee.

Nutritional status was classified according to Weight for Age (IAP Classification of Malnutrition, IAP and WHO growth charts), Height for Age (Waterlows Classification of Stunting, IAP and WHO growth Charts), Weight For Height (Water lows Classification of Wasting, IAP and WHO growth Charts) and Mid Upper Arm Circumference, Body Mass Index(IAP and WHO Charts)

Table 2: IAP Classification of Underweight:[63]

Nutritional status	Weight for age
Normal	>80%
Grade I (Mild)	71-80%
Grade II (Moderate)	61-70%
Grade III (Severe)	51-70%
Grade IV (Very Severe)	<50%

Table 3: Waterlows Classification of Stunting:[63]

Grading	Height for age
Normal	>95%
First Degree (Mild)	90-95%
Second Degree (Moderate)	85-90%
Third Degree (Severe)	<80

Table 4: Waterlows Classification of Wasting:[63]

Grading	Weight for height
Normal	>90%
First Degree (Mild)	80-90%
Second Degree (Moderate)	70-80%
Third Degree (Severe)	<70%

Midupper Arm Circumference (6-60 Months):[63]

- ✓ >13.5 cm: Normal
- ✓ 12.5-13.5: Moderate Malnutrition
- ✓ <12.5: Severe Malnutrition
- ✓ <11.5: SAM (Severe Acute Malnutrition)

Table 5: WHO Hemoglobin levels to Diagnose Anemia at Sea level (gm/l), 2016:[64]

Population	Non Anemia	Anemia		
		Mild*	Moderate	Severe
Children 6-59 months of age	110 or higher	100-109	70-99	lower than 70
Children 5-11 years of age	115 or higher	110-114	80-109	lower than 80
Children 12-14 years of age	120 or higher	110-119	80-109	lower than 80
Non-pregnant women (15 years of age and above)	120 or higher	110-119	80-109	lower than 80
Pregnant women	110 or higher	100-109	70-99	lower than 70
Men (15 years of age and above)	130 or higher	110-129	80-109	lower than 80

*'Mild' is a misnomer; iron deficiency anemia is already advanced by the time anemia is detected. The deficiency has consequences even when no anemia is clinically apparent

WHO Diagnostic Criteria Polycythemia:[65]

- Hemoglobinb>18.5 g/dl (men)
- Hemoglobin >16.5g/dl(women)

Table 6: Normal Red Cell Indices:[66]

Age Group	Red Cell Indices		
	MCV (fL)	MCH(pg)	MCHC(gm/dl)
1-23 months	72-88	24-30	32-36
2-9 years	76-90	25-31	
10- 17 years	78-95	26-32	

- RDW(%)[66] :11.5-14.5

STATISTICAL AND DATA ANALYSIS:

The data was entered into Microsoft excel sheet 2007 and was analysed using appropriate package of SPSS Version 20. Summary statistics was assessed using descriptive analysis. Statistical significance was assessed using Chi square test. A p value of <0.05 was considered significant.

RESULTS

A total of 80 children with congenital heart disease who satisfied the inclusion criteria were enrolled in this study

Table 7: Age Distribution

AGE	No.	%
12-59 months	29	36.2
5-12 years	51	63.8
Total	**80**	**100**

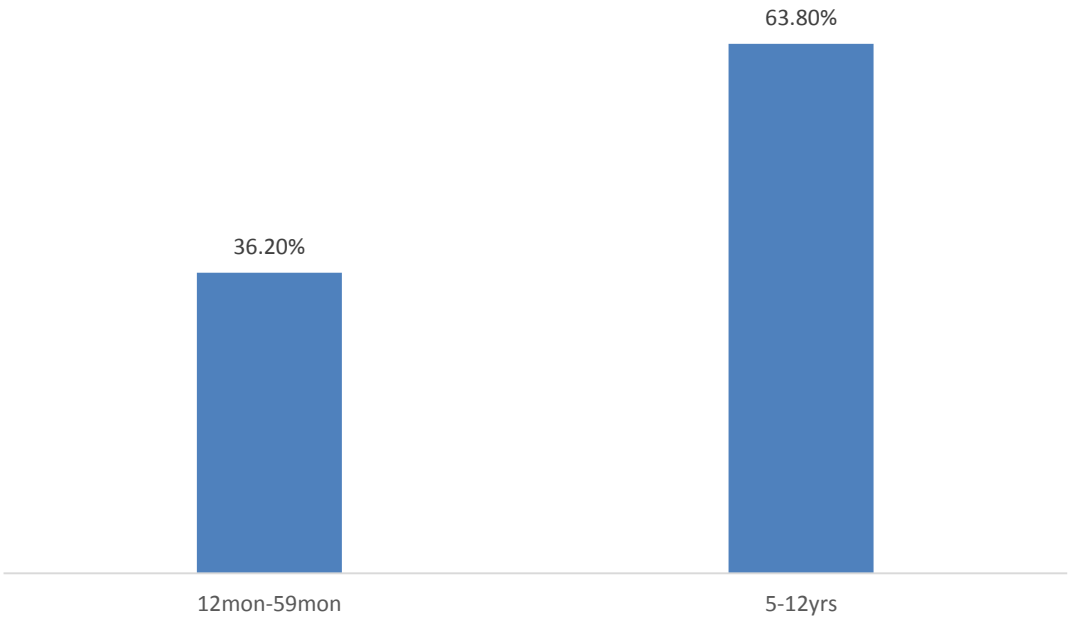


Fig. 4: Distribution according to age

In the present study, 36.2% belonged to 12mon-59mon and 63.8% belonged to 5-12years

Table 8: Sex Distribution

SEX	No	%
Male	40	50
Female	40	50
Total	**80**	**100**

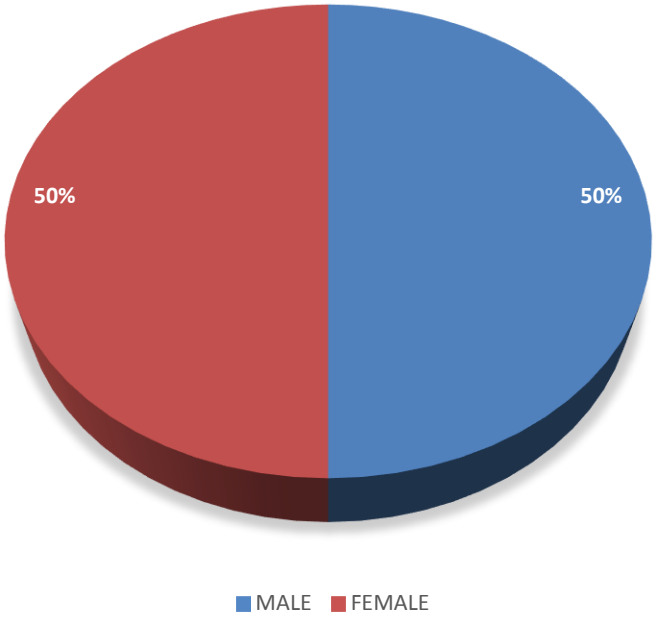


Fig. 5: Distribution according to Sex

In this study, 50% were male and 50% were female. Male to female ratio was 1:1.

Table 9: Distribution according to Age and Sex:

AGE	SEX				Total	
	Male		Female			
	N	%	N	%	N	%
12-59 months	14	48.3	15	51.7	29	100
5-12 years	26	51	25	49	51	100
Total	40		40		80	100

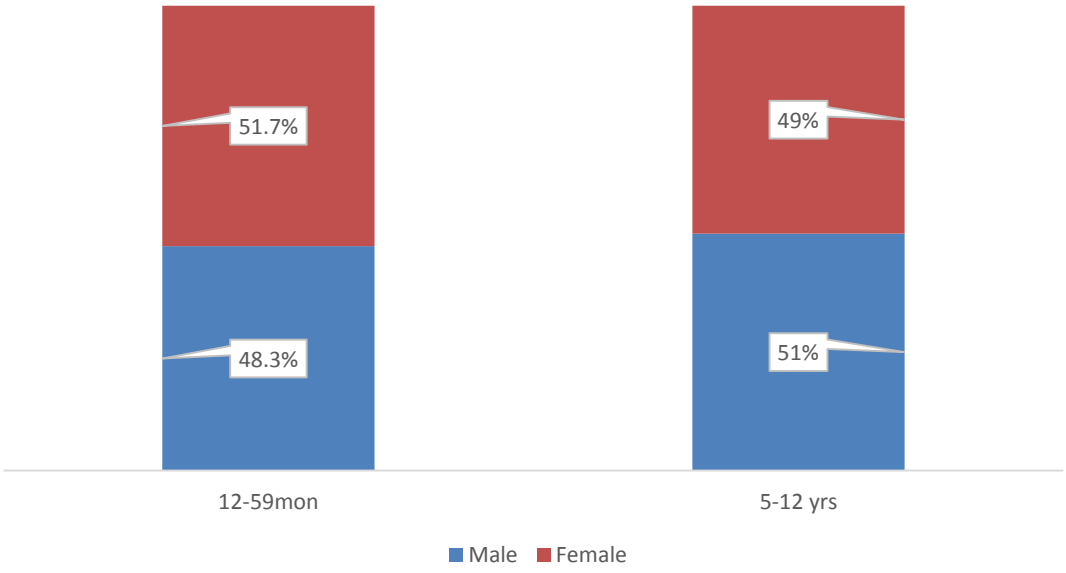


Fig.6: Distribution according to age and sex

Comparing the distribution of age group and sex, out of the 29 children in the age group 12-59 moths, 48.3% are male and 51.7% are female whereas out %1 children in the age group 5-12yrs, 51% are female and 49% are male.

Table 10: Distribution According to type of Congenital Heart Disease (n=80)

TYPE		No.	%
ACYANOTIC	VSD	28	35
	ASD	24	30
	PDA	8	10
	OTHERS*	5	6.3
Total ACHD		65	81.3
CYANOTIC	TOF	11	13.7
	OTHERS**	4	5
Total CCHD		15	18.7

OTHERS*→Pulmonary stenosis, Aortic Stenosis, Coarctation of Aorta

OTHERS**→Complex Heart Disease, Double outlet right Ventricle,

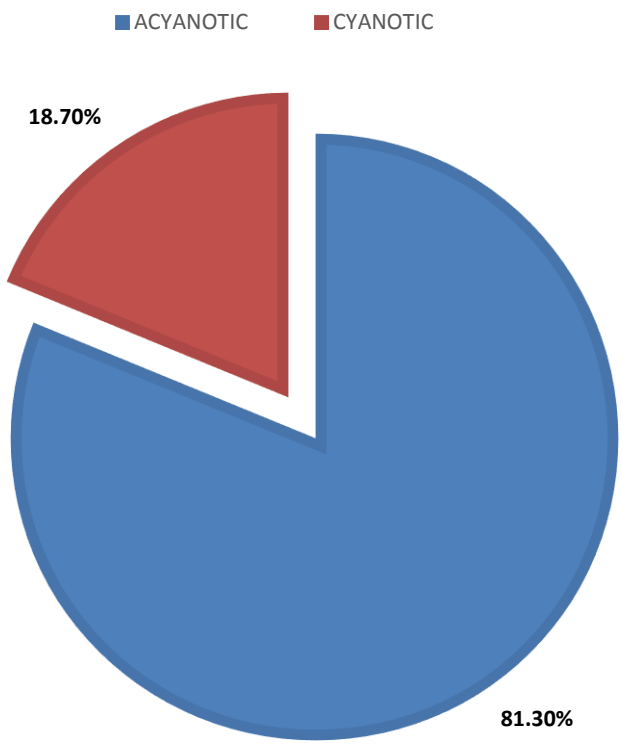


Fig.7: Distribution according to type of congenital heart disease

Out of the 80 children enrolled with Congenital Heart Disease, 81.2% had acyanotic congenital heart disease and 18.8% had cyanotic congenital heart disease

In ACHD group, VSD(35%) was more predominant followed by ASD(30%) and PDA(10%) and Others(6.3%)

In CCHD group, TOF(13.7%) was more predominat and others(5%)

Table 11: Distribution according to Socio Economic Status

SOCIO ECONOMIC STATUS	No.	%
Upper Class (I)	-	-
Upper Middle Class (II)	3	3.8
Lower Middle Class (III)	7	8.8
Upper Lower Class (IV)	53	66.2
Lower Class (V)	17	21.2
Total	80	100

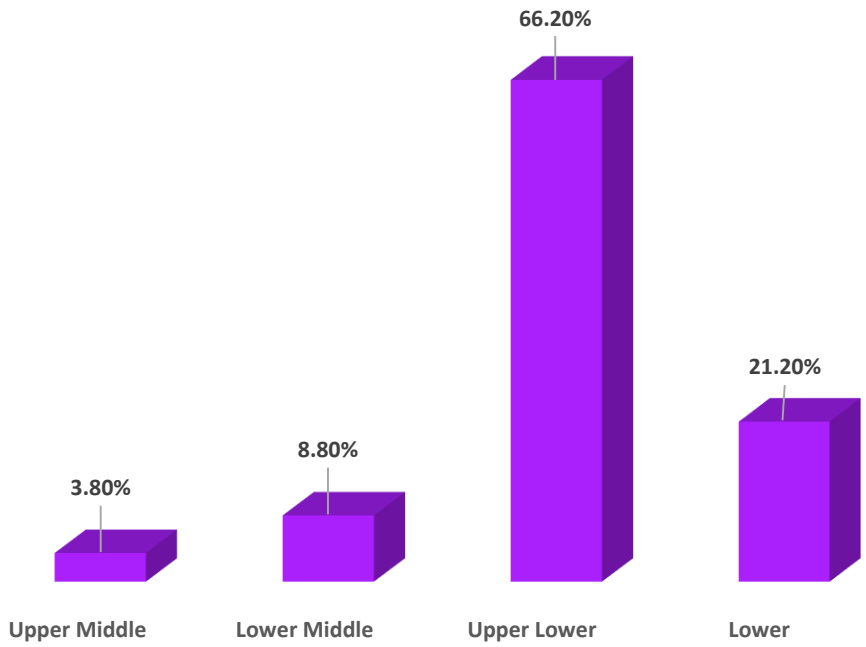


Fig.8: Distribution according to Socio economic status

In this present study, majority of the children with congenital heart disease belonged to lower(87.4%) class with Upper lower class(66.2%) and lower class(21.2%)

Table 12: Distribution according to family members affected

FAMILY MEMBERS AFFECTED	No.	%
Yes	1	1.2
No	79	98.8
Total	80	100

In this present study, only 1.2% of the family member (paternal uncle) had a history of congenital heart disease (acyanotic-VSD) corresponding to the lesion in the affected child for which surgical correction was done

Table 13: Distribution According to Clinical Features of Malnutrition

Clinical features of malnutrition considered were clinical pallor, Loose skin folds, Bipedal edema, Vitamin A Deficiency, Vitamin B Complex Deficiency, Skin changes, Hair Changes, Apathy)

None of the children had any features of SAM

CLINICAL FEATURES		No.	%
Pallor	No Pallor	60	75
	Pallor	20	25
Vitamin A Deficiency - Bitots Spots		3	3.8
Vitamin B Complex Deficiency-Angular stomatitis		4	5
Vitamin D Deficiency-Frontal bossing, Rachitic rosary		3	3.8

In this study, only 25% had pallor had 5% had Vitamin B complex deficiency, 3.8% had Vitamin A and Vitamin D deficiency.

Table 14: Distribution according to Maternal Age at time of conception

MATERNAL AGE	No.	%
<19 years	5	6.2
19- 35years	74	92.5
>35years	1	1.2
Total	80	100

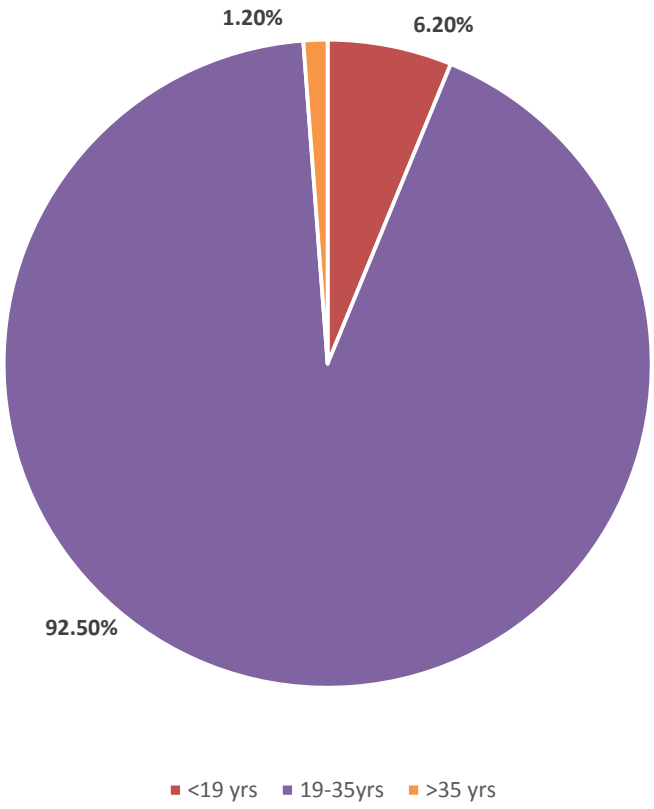


Fig.9: Distribution according to maternal age at time of conception

In this study, 6.2% of the mothers were below 19 years, 92.5% belonged to 19-35years and 1.2% were above 35 years at the time of conception

Table 15: Distribution according to Degree of Consanguinity

CONSANGUINITY(DEGREE)	No.	%
First	-	-
Second	50	62.5
Third	18	22.5
Total	80	100

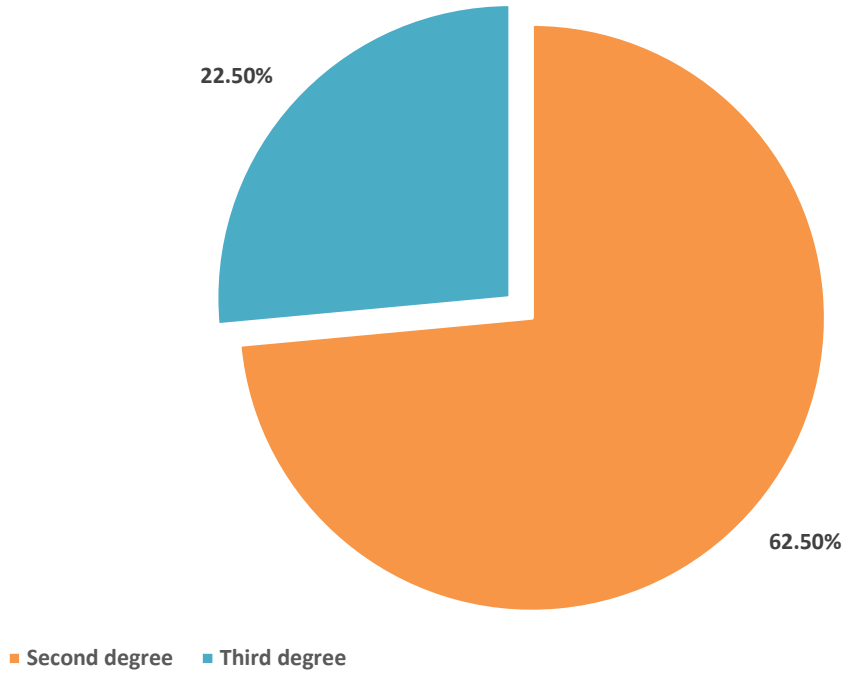


Fig.10: Distribution according to degree of consanguinity

Majority of children with congenital heart disease had parents who were second degree (62.5%) consanguinity and only 22.5% were third degree consanguinity

Table 16: Distribution according to Birth Order

BIRTH ORDER	No.	%
First	25	31.2
Second	45	56.2
Third	10	12.5
Total	80	100

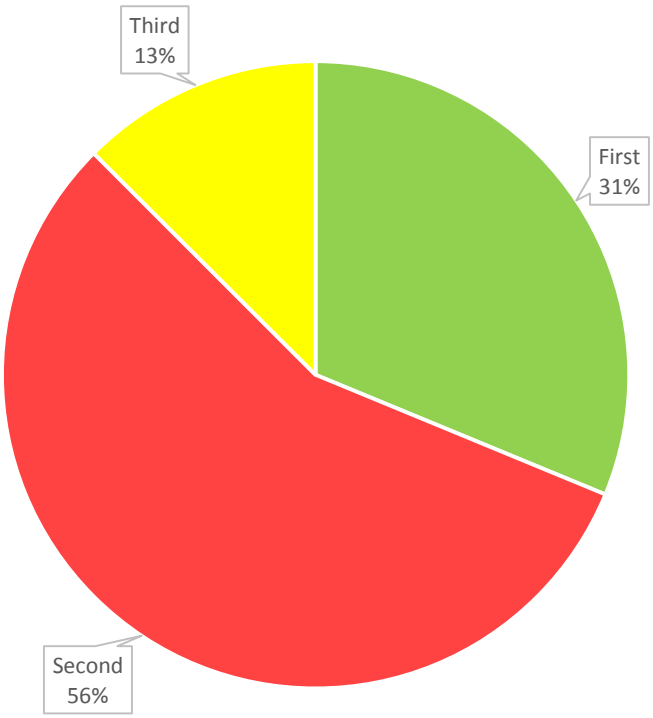


Fig.11: Distribution according to birth order

In this study, majority of the children with the congenital heart disease belonged to second birth order (56.2%) whereas 31.2% belonged to first and 12.5% third birth order.

Table 17: Distribution according to Gestational Age

GESTATION	No.	%
Preterm	5	6.2
Term	75	93.8
Total	80	100

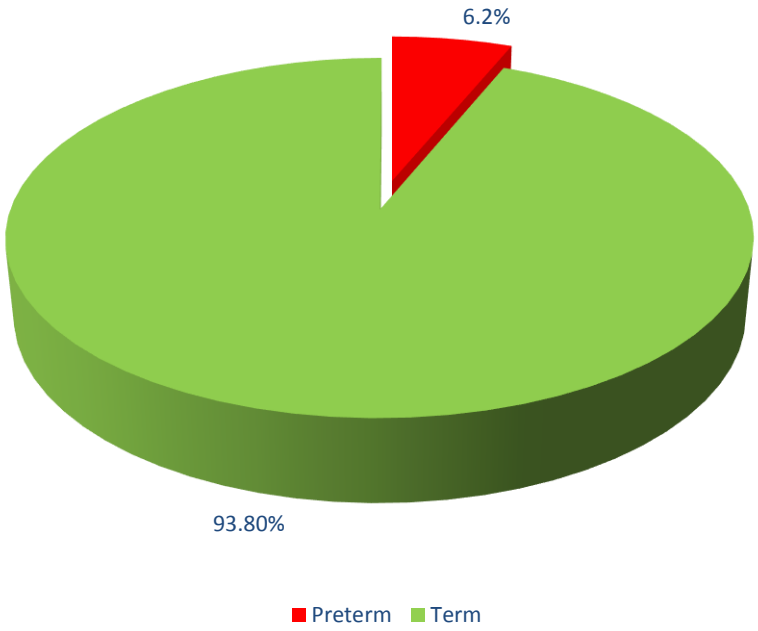


Fig.12: Distribution according to gestational age

In this study only 6.2% were preterm (<37weeks) and 93.8% were term(>37 weeks)

TABLE 18: Distribution according to Birth Weight

BIRTHWEIGHT	No.	%
Normal	66	82.5
Low Birth Weight	14	17.5
Total	80	100

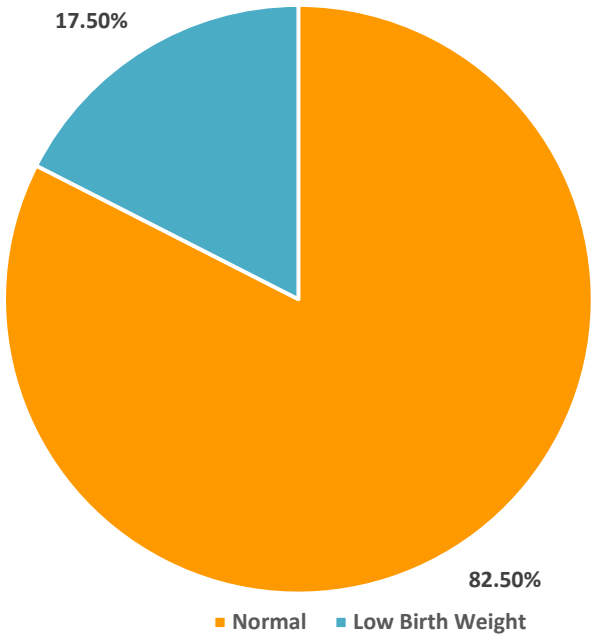


Fig.13: Distribution according to birth weight

Only 17.5% out of the total children were low birth weight(<2.5kg) and 82.5% had normal birth weight(2.5-3kg)

Table 19: Distribution according to IYCF Practices (n=80):

EXCLUSIVE BREAST FEEDING TILL 6 MONTHS	**No.**	**%**
Yes	66	82.5
No	14	17.5
BREASTFEEDING TILL 2 YRS	**No.**	**%**
Yes	27	33.8
No	53	66.2
COMPLEMENTARY FEEDING AT END OF 6MON	**No.**	**%**
Yes	60	75
No	20	25
FAMILY POT FEEDING BY 1 YEAR OF AGE	**No.**	**%**
Yes	74	92.5
No	6	7.5

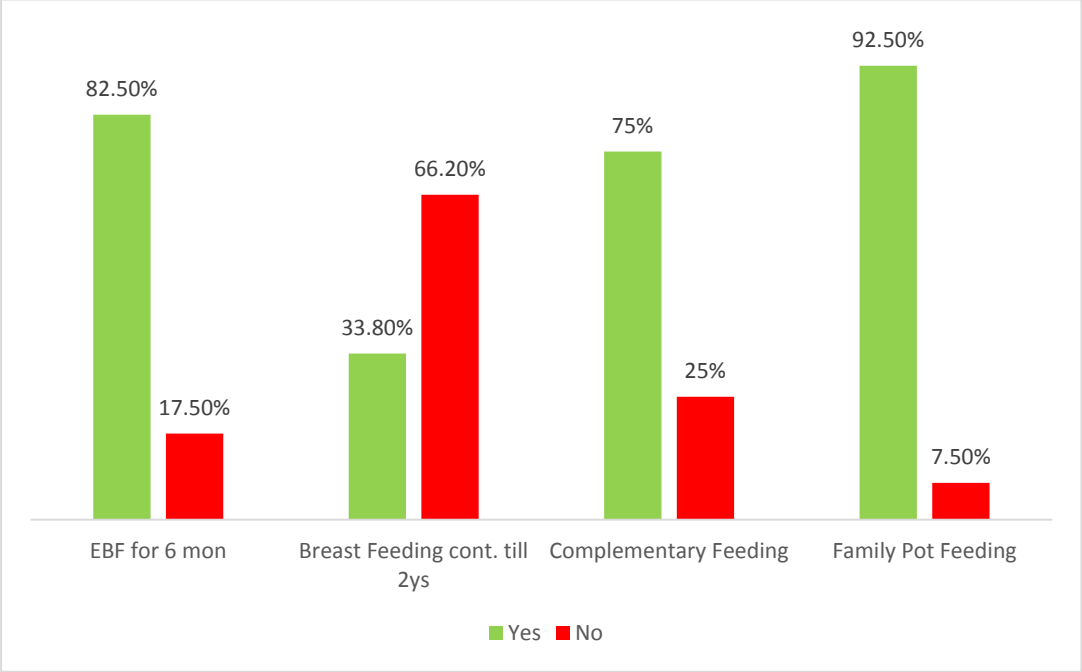


Fig.14: Distribution according to IYCF practices

Majority of the children (82.5%) were exclusively breast fed till 6 months Only 33.8% were continued breast feeding upto 2 years whereas 66.2% were not.

Majority of the children (75%) were started on complementary feeding in the form of mashed rice, mashed idli by end of 6 months

Most of the children (92.5%) were started on family pot feeding by end of 1 year of age

Table 20: Distribution according to Dietary Intake (n=80)

CALORIE INTAKE	No.	%
<50%	4	5
50-75%	75	93.8
76-90%	1	1.2
>90%	-	-

PROTEIN INTAKE	No.	%
<50%	4	5
50-75%	75	93.8
76-90%	1	1.2
>90%	-	-

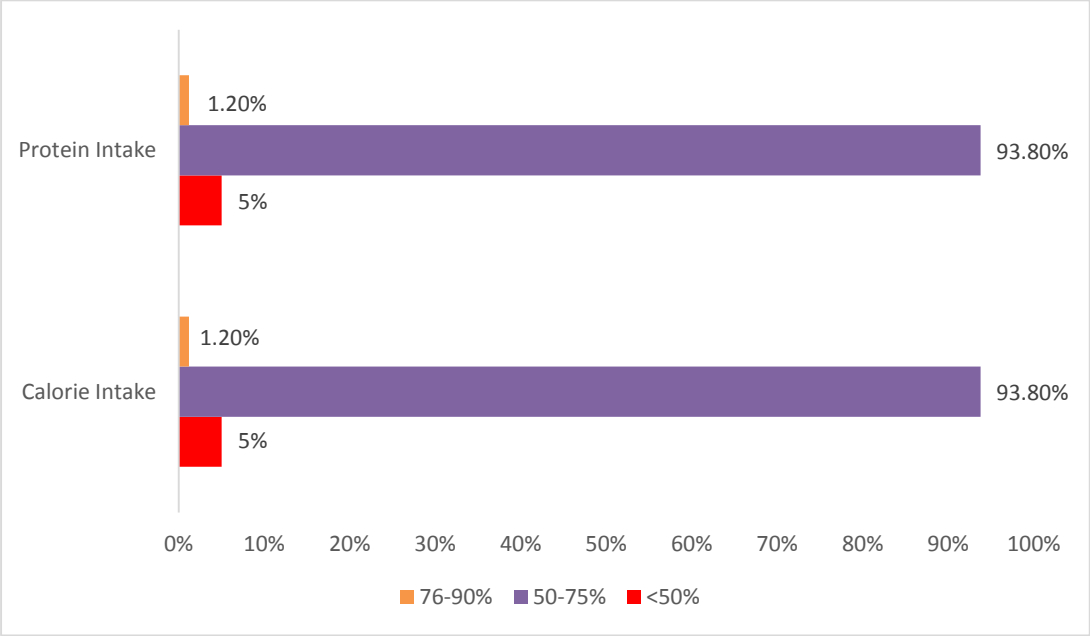


Fig.15: Distribution according to dietary intake

In this study, 5% of the children took <50% of the required calorie and proteins,93.8% took 50-75% of the required calorie and proteins and only 1.2% took 76-90% of the required calories and protein

NUTRITIONAL STATUS (WFA, HFA, WFH, BMI, MUAC) (N=80)

Table 21: Distribution according to Weight for Age(WfA)

WfA	No.	%
Normal(>80%)	20	25
Total Underweight(<80%)	60	75
Mild(71-80%)	22	27.5
Moderate(61-70%)	24	30
Severe (51-60%)	13	16.2
Very Severe(<50%)	1	1.2
Total	80	100

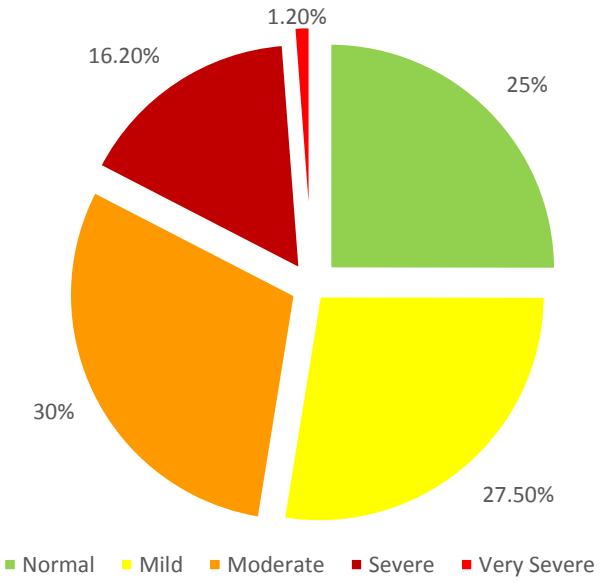


Fig.16: Distribution according to weight for age

In this present study, according to WfA,25% were normal and 75% were underweight.

27.5% had mild, 30% had moderate,16.2% had severe and 1.2% had very severe underweight.

Table 22: Comparison between ACHD and CCHD according to Weight for Age

Weight for Age (WfA)	Type of Congenital Heart Disease				Total	
	Acyanotic		Cyanotic			
	N	%	N	%	N	%
Normal	19	95	1	5	20	100
Mild Underweight	20	90.9	2	9.1	22	100
Moderate Underweight	17	70.8	7	29.2	24	100
Severe Underweight	9	69.2	5	35.7	14	100
Very severe Underweight	0	0	0	0	0	0
Total					80	100

χ^2 : 11.10 ; p-value : 0.025; Statistically significant

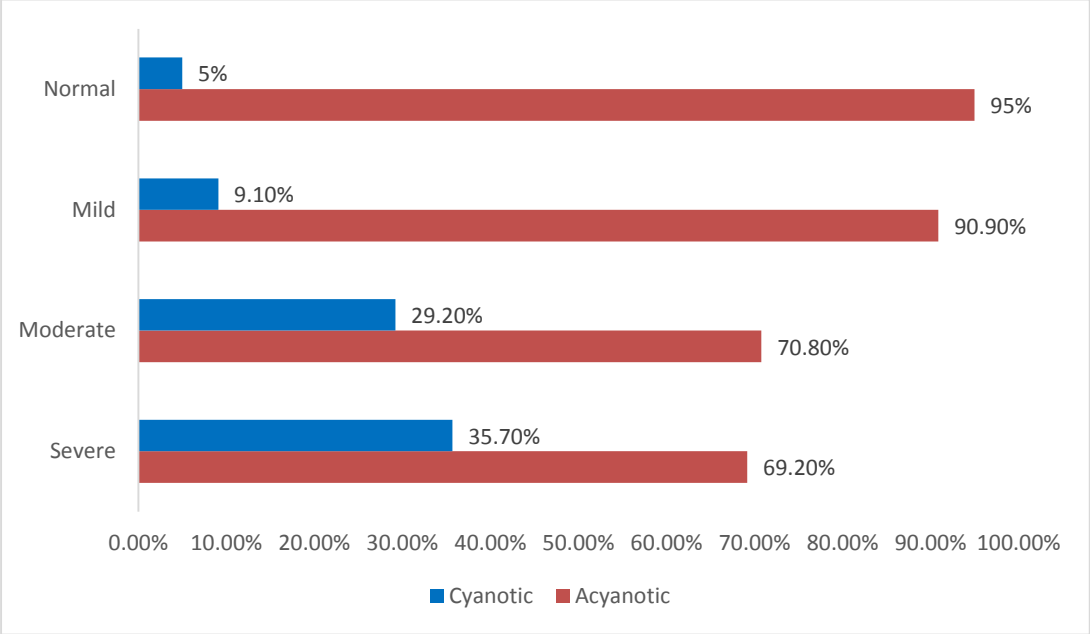


Fig 17: Comparison between ACHD and CCHD according to weight for age

In children with ACHD, 90.9% had had mild, 70.8% had moderate,69.2% had severe underweight according to weight for age whereas in children with CCHD ,9.1% had mild,29.2% had moderate and 35.7% had severe underweight. Statistically significant with p value - 0.025

Table 23: Comparison between age according to Weight for Age

Weight For Age (WfA)	Age				Total	
	12-59 months		5-12yrs			
	N	%	N	%	N	%
Normal	9	45	11	55	20	100
Mild underweight	10	4.5	12	54.5	22	100
Moderate underweight	6	25	18	75	24	100
Severe underweight	4	28.5	10	71.4	14	100
Very Severe underweight	-	-	-	-	-	-
Total	29		51		80	100

χ^2 : 5.518 ; p-value : 0.238; Statistically not significant

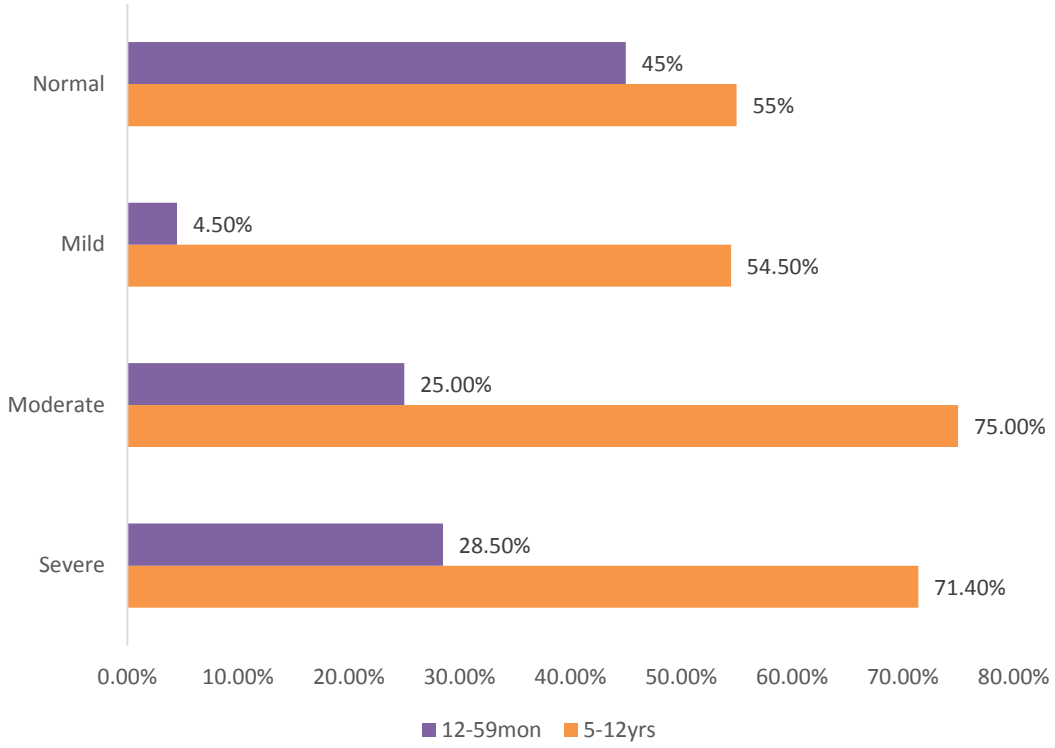


Fig.18: Comparison between age according to weight for age

Underweight was more common in children aged 5-12 years (mild-54.5%,moderate-75%,severe-71.4%) compared to children between 12-59mon(mild-4.5%,moderate-25%,severe-28.5%)

TABLE 24: Distribution according to Height for Age(HfA):

HfA	No.	%
Normal(>95%)	34	42.5
Total Stunting	46	57.5
Mild(90-95%)	28	35
Moderate(85-90%)	11	13.8
Severe (<85%)	7	8.8
Total	80	100

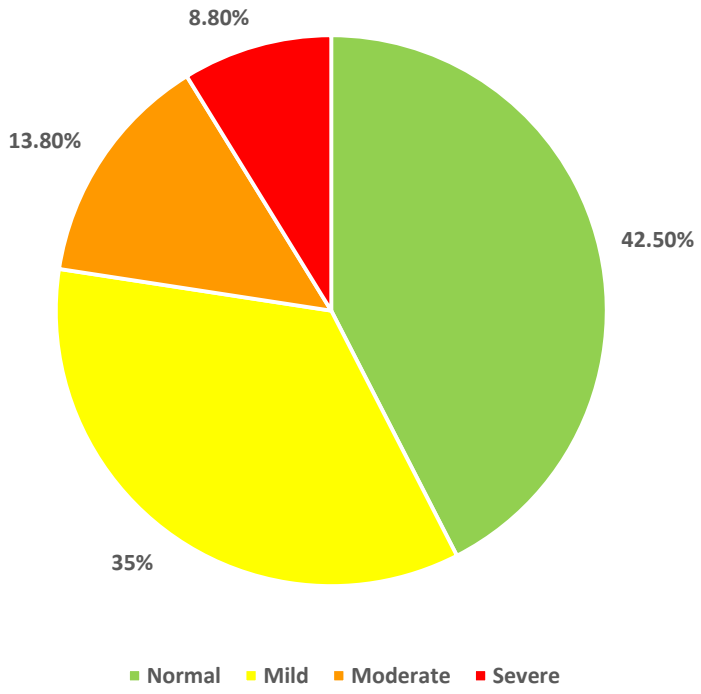


Fig.19: Distribution according to height for age

In this present study, 42.5% were normal and 57.5% were stunted according to height for age.

35% had mild, 13.8% had moderate, 8.8% had severe stunting.

Table 25: Comparison between ACHD and CCHD according to Height for Age

Height for Age(HfA)	Type of Congenital Heart Disease				Total	
	Acyanotic		Cyanotic			
	N	%	N	%	N	%
Normal	33	97.1	1	2.9	34	100
Mild stunting	21	75	7	25	28	100
Moderate stunting	8	72.7	3	27.3	11	100
Severe stunting	3	42.9	4	57.1	7	100
Total					80	100

χ^2 : 13.6; p-value : 0.004; Statistically significant

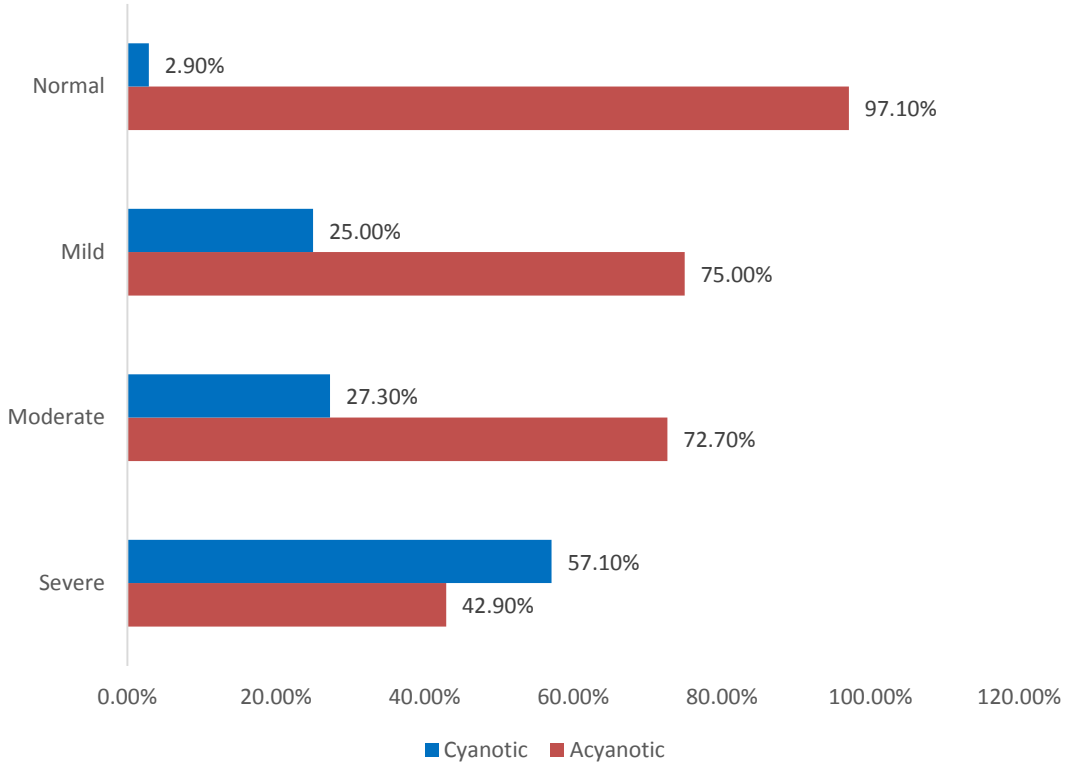


Fig.20: Comparison between ACHD and CCHD according to height for age

In children with ACHD, 75% had mild,72.7% had moderate,42.9% had severe stunting according to height for age whereas in childern with CCHD ,25% had mild,27.3% had moderate,57.1% had severe stunting,. Statistically significant with p value - 0.004

Table 26: Comparison between age according to Height for Age

Height for Age (HfA)	Age				Total	
	12-59 months		5-12yrs			
	N	%	N	%	N	%
Normal	10	29.4	24	70.6	34	100
Mild stunting	10	35.7	18	64.3	28	100
Moderate stunting	4	36.4	7	63.6	11	100
Severe stunting	5	71.4	2	28.6	7	100
Total					80	100

χ^2 : 4.44 ; p-value : 0.218; Statistically not significant

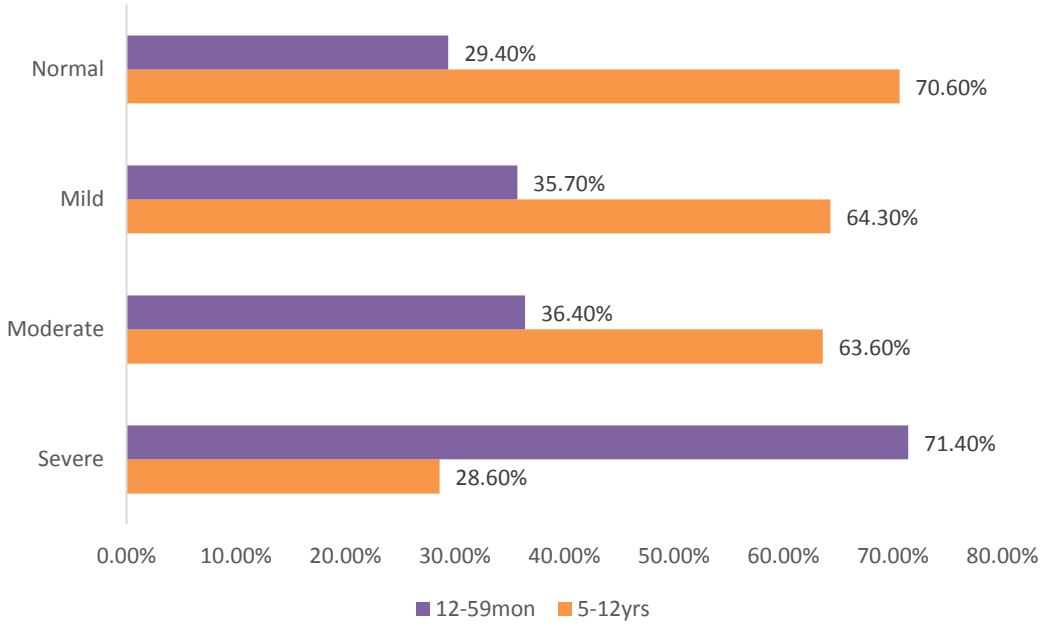


Fig.21: Comparison between age according to weight for age

Mild and Moderate stunting was more common in children aged 5-12 years -64.3% and 63.6% respectively compared to children with 12-59months-35.7% and 36.4% respectively but severe stunting was common in age group 12-59 months compared to 5-12yrs (71.4% vs 28.6%).

Table 27: Distribution according to Weight for Height (WfH):

WfH	No.	%
Normal(>90%)	13	16.2
Total Wasting	67	83.7
Mild(80-90%)	35	43.8
Moderate(70-80%)	22	27.5
Severe (<70%)	10	12.5
Total	80	100

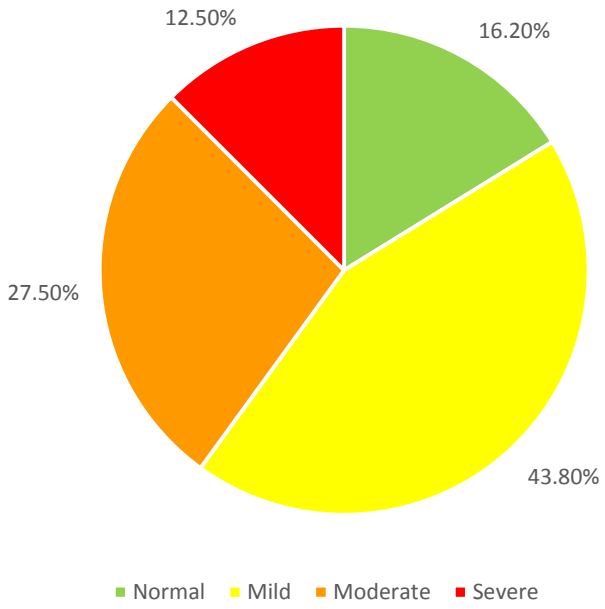


Fig.22: Distribution according to weight for height

In this study, 16.2% were normal and 83.7% were wasted according to weight for height, 43.8% had mild, 27.5% had moderate and 12.5% had severe wasting.

Table 28: Comparison between ACHD and CCHD according to Weight for Height

Weight for Height(WfH)	Type of Congenital Heart Disease				Total	
	Acyanotic		Cyanotic			
	N	%	N	%	N	%
Normal	12	92.3	1	7.7	13	100
Mild wasting	31	88.6	4	11.4	35	100
Moderate wasting	14	63.6	8	36.4	22	100
Severe wasting	8	80	2	20	10	100
Total					80	100

χ^2 : 6.76; p-value : 0.080; Statistically not significant

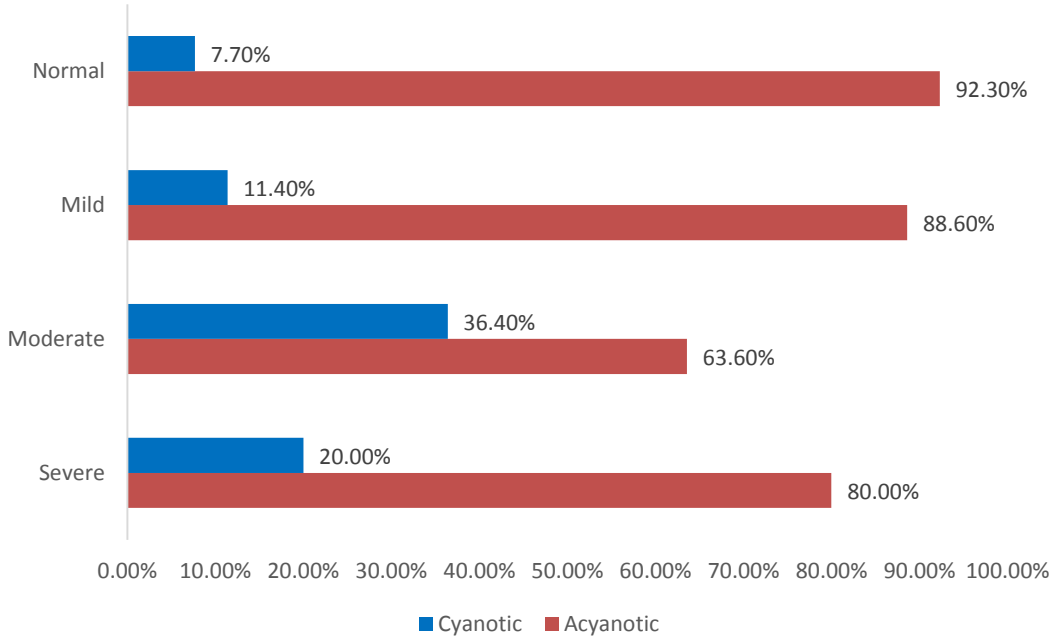


Fig.23: Comparison between ACHD and CCHD according to weight for height

In children with ACHD, 88.6% had mild, 63.6% had moderate, 80% had severe wasting according to weight for height whereas in children with CCHD, 11.4% had mild,36.4%had moderate, 20% had severe wasting . Statistically not significant.

Table 29: Comparison between age according to Weight for Height

Weight For Height (HfA)	Age				Total	
	12-59 months		5-12yrs			
	N	%	N	%	N	%
Normal	6	46.2	7	53.8	13	100
Mild wasting	11	31.4	24	68.6	35	100
Moderate wasting	8	36.4	14	63.6	22	100
Severe wasting	4	40	6	60	10	100
Total					80	100

χ^2 : 0.965; p-value : 0.810; Statistically not significant

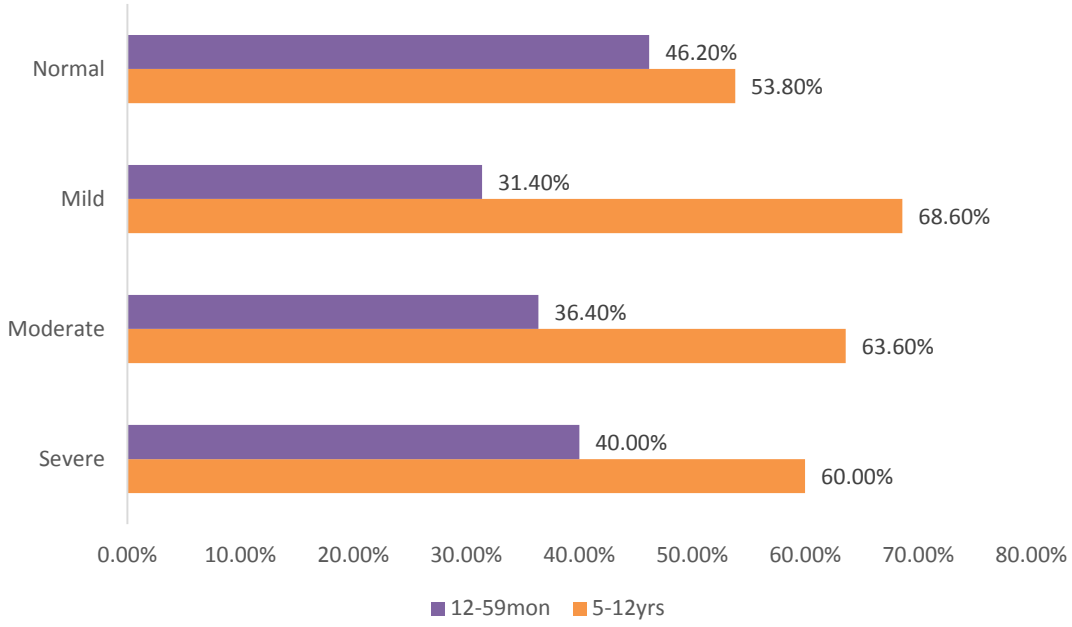


Fig.24: Comparison between age according to weight for height

Wasting was more common in children aged 5-12 years (mild - 68.6%, moderate - 63.6%, severe - 60%) compared to children between 12-59 mon (mild-31.4%, moderate - 36.4%, severe - 40%)

TABLE 30: Distribution according to BMI:

BMI	No.	%
Normal	13	16.2
Total CED*	67	83.8
Moderate CED	49	61.2
Severe CED	18	22.5
Total	80	100

*CED - Chronic Energy Deficiency

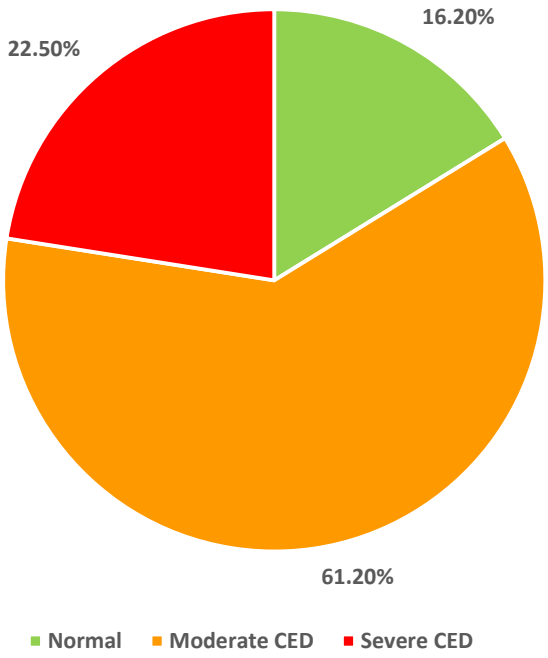


Fig.25: Distribution according to BMI

In this study, 83.7% had chronic energy deficiency (CED).

Majority (61.2%) had moderate chronic energy deficiency and 22.5% had severe chronic energy deficiency

Table 31: Comparison between ACHD and CCHD according to BMI

BMI	Type of Congenital Heart Disease				Total	
	Acyanotic		Cyanotic			
	N	%	N	%	N	%
Normal	11	84.6	2	15.4	13	100
Moderate CED	43	87.8	6	12.2	49	100
Severe CED	10	55.5	8	44.5	18	100
Total					80	100

χ^2 : 7.3; p-value : 0.063; Statistically not significant

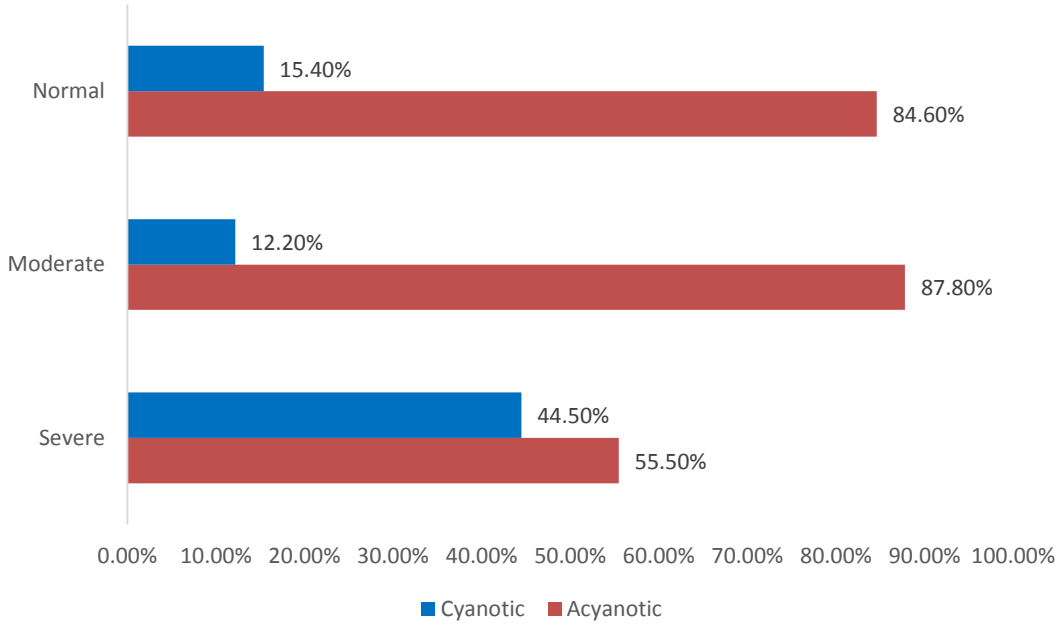


Fig.26: Comparison between ACHD and CCHD according to BMI

In children with ACHD, 87.8% had moderate and 55.5% had severe CED according to BMI, whereas in children with CCHD, 12.2% h ad moderate and 44.5% had severe CED. Statistically not significant

Table 32: Comparison between age according to BMI

BMI	Age				Total	
	12-59 months		5-12yrs			
	N	%	N	%	N	%
Normal	5	38.5	8	61.5	13	100
Moderate CED	19	38.8	30	61.2	49	100
Severe CED	5	27.7	13	72.3	18	100
Total					80	100

χ^2 : 1.07; p-value : 0.78; Statistically not significant

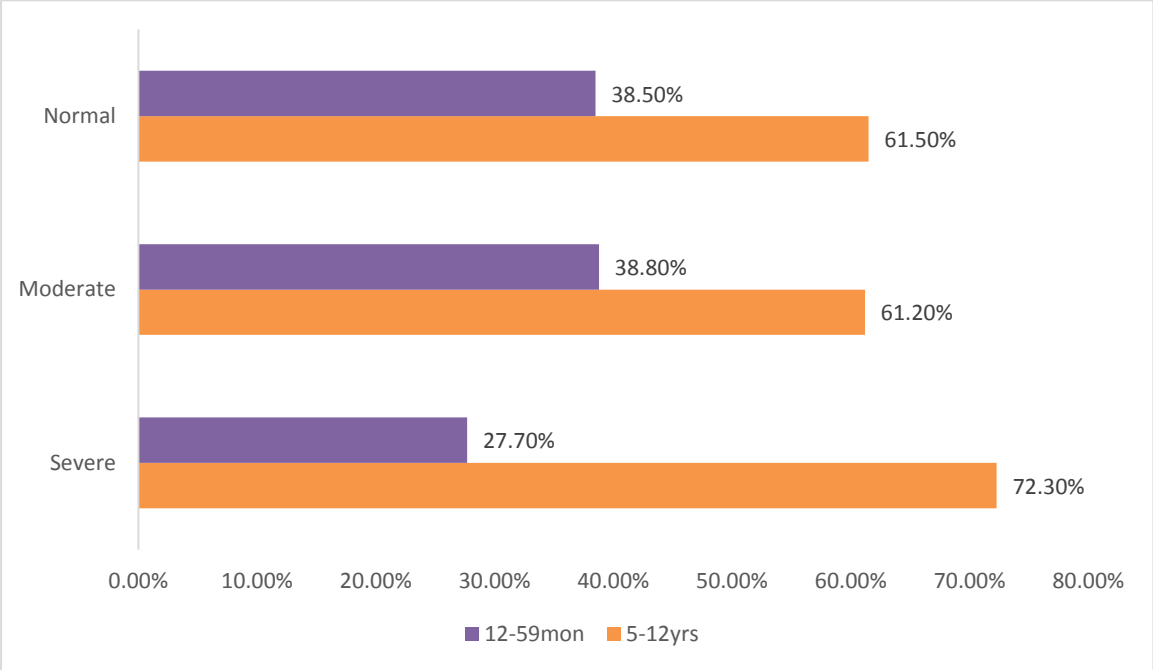


Fig.27: Comparison between age according to BMI

Chronic energy deficiency based on BMI was more common in children aged 5-12 years (moderate - 61.2 %, severe - 72.3%) compared to children between 12-59 mon (moderate - 38.8%, severe - 27.7%)

Table 33: Distribution According To MUAC (6-59 Months)

MUAC	No.	%
Normal(>13.5cm)	17	51.5
Malnutrition(<13.5cm)	14	42.4
Mild-Moderate(12.5cm-13.5cm)	9	27.2
Severe (<12.5cm)	5*	15.1
Total	33	100

*Out of the 5 with severe malnutrition,, 3 had <11.5cm classified as Severe acute malnutrition(SAM)

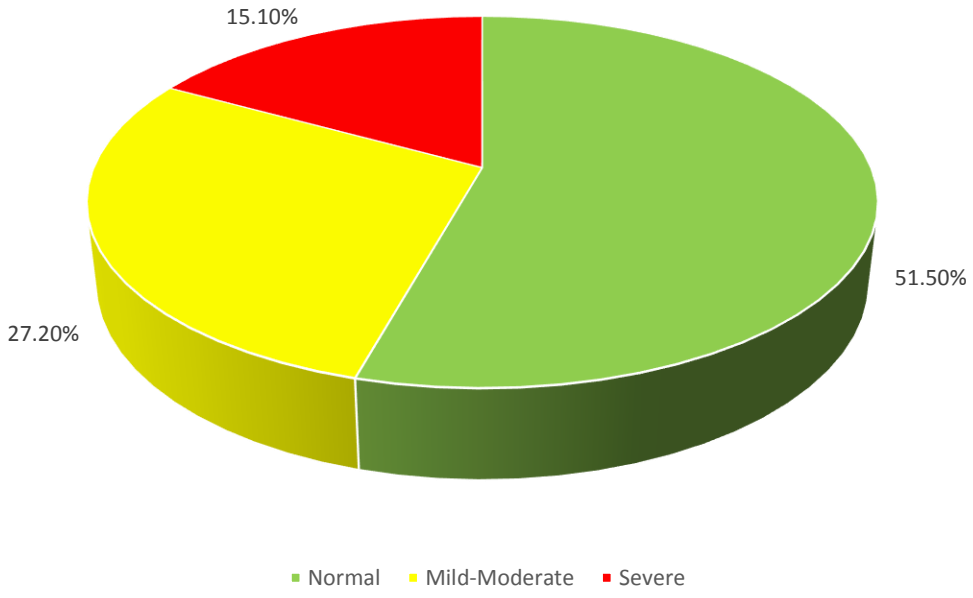


Fig.28: Distribution according to MUAC

Only 33 children were in the age group of 6-59 months,According to MUAC,51.5% were normal(>13.5cm) and 42.4% were malnourished(27.2% had mild to moderate malnutrition and 15.1% had severe malnutrition)

DEGREE OF ANEMIA BASED ON HEAMOGLOBIN, RED CELL INDICIES, RDW AND PERIPHERAL SMEAR:

Table 34: Distribution according to Heamoglobin(gm/dl):

HEAMOGLOBIN(gm/dl)	No.	%
Normal	49	61.3
Anemia	17	21.3
Mild Anemia	15	18.8
Moderate Anemia	2	2.5
Severe Anemia	-	-
Polycythemia	14	17.5
TOTAL	80	100

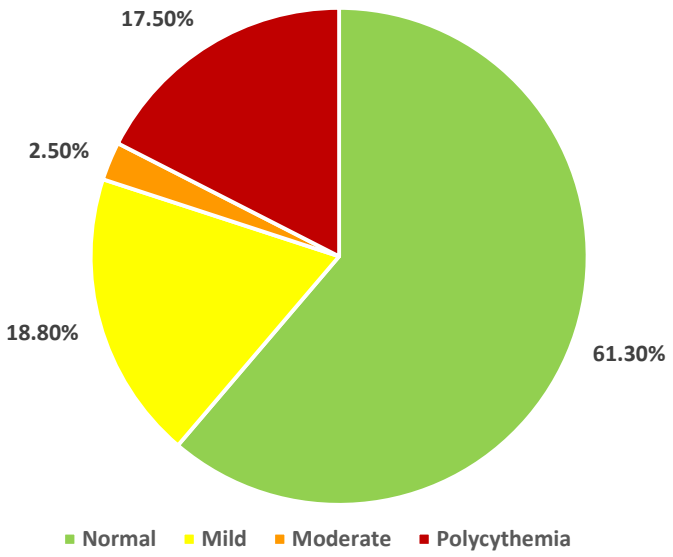


Fig. 29: Distribution according to hemoglobin estimation

In this study, as per hemoglobin estimations and grading anemia according to WHO guidelines,61.3% of the children with congenital heart disease were normal and 21.3% had anemia (18.8% had mild anemia,2.5% had moderate anemia).17.5% had polycythemia. Hemoglobin is not a sensitive marker in CCHD.

TABLE 35: Distribution according to Red Cell Indices (MCV, MCH and MCHC):

RED CELL INDICES MCV(fL),MCH(pg),MCHC(gm/dl)	No.	%
Normal	63	78.8
Decreased	17	21.2
Total	80	100

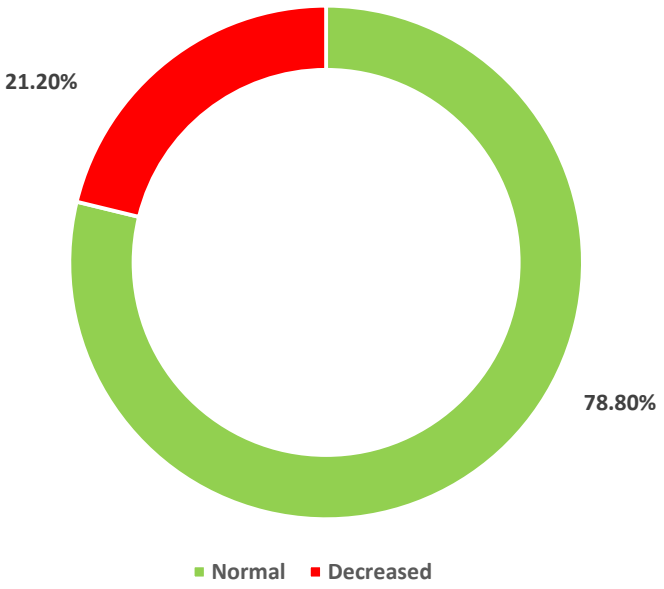


Fig. 30: Distribution according to Red cell indices

In this study, 21.2% had all decreased red cell indices and rest (78.8%) had normal red cell indices

Table 36: Comparison between ACHD and CCHD according to Red Cell Indices

Red Cell Indices MCV(fL), MCH(pg), MCHC(gm/dl)	Type of Congenital Heart Disease				Total	
	Acyanotic		Cyanotic			
	N	%	N	%	N	%
Normal	52	82.5	11	17.5	63	100
Decreased	13	76.5	4	23.5	17	100

χ^2 : 0.32;p-value : 0.56; Statistically not significant

In children with ACHD, 82.5% had normal red cell indices and 76.5% had decreased red cell indices vs children with CCHD, 17.5% had normal red cell indices and 23.5% had decreased red cell indices

Table 37: Distribution according to RDW:

RDW (%)	No.	%
Normal	51	63.8
Increased	29	36.2
Total	80	100

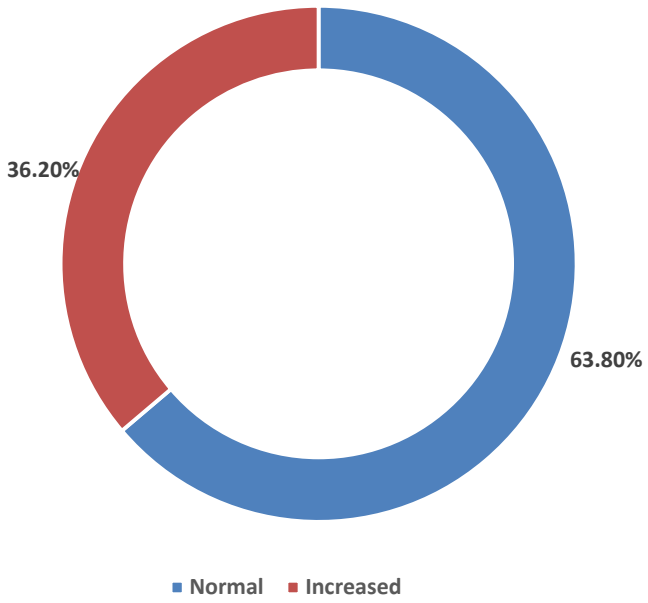


Fig. 31: Distribution according to RDW

In this study 36.2% had increased RDW indicating nutritional deficiency and 51% had normal RDW

Table 38: Comparison between ACHD and CCHD according to RDW

RDW(%)	Type of Congenital Heart Disease				Total	
	Acyanotic		Cyanotic			
	N	%	N	%	N	%
Normal	49	96.1	2	3.9	51	100
Increased	16	55.2	13	44.8	29	100

χ^2 : 20.3;p-value : 0.000; Statistically significant

In children with ACHD, 96.1% had normal RDW and only 55.2% had increased RDW whereas children with CCHD, 3.9% had normal RDW and 44.8% had increased RDW. Statistically significant with p value 0.000

TABLE 39: Distribution according to Peripheral Smear

PERIPHERAL SMEAR	No.	%
Microcytic Hypochromic	13	16.2
Normocytic Normochromic	67	83.8
Total	80	100

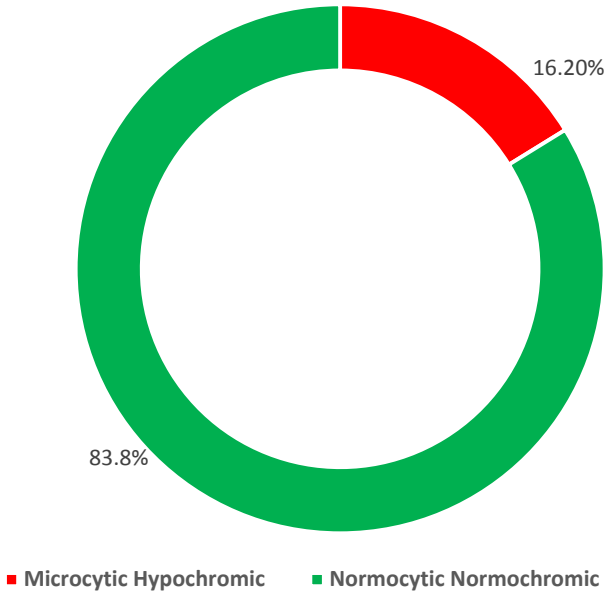


Fig. 32: Distribution according to peripheral smear

16.2% had microcytic hypochromic anemia and the rest (83.8%) had normocytic normochromic anemia.

Table 40: Comparison between ACHD and CCHD according to Peripheral Smear

PERIPHERAL SMEAR	Type of Congenital Heart Disease				Total	
	Acyanotic		Cyanotic			
	N	%	N	%	N	%
Microcytic Hypochromic	10	76.9	3	23.1	13	100
Normocytic Normochromic	55	82.1	12	17.9	67	100

χ^2 : 0.19;p-value : 0.622 Statistically not significant

In children with ACHD, only 76.9.% had microcytic hypochromic and rest had normocytic normochromic whereas children with CCHD, 23.1% had microcytic hypochromic and the rest had normocytic normochromic

DISCUSSION

Congenital heart defect (CHD) is the most common congenital malformation among all birth defects leading to morbidity and mortality among children. The burden of CHD is high in developing countries like India, due to the high birth rate and critical nature of CHD requiring expensive surgical and non-surgical interventions

Congenital heart disease (Cyanotic and Acyanotic) occurs in approximately 0.8% of live births worldwide. In India, the prevalence of CHD is not uniform across the country and varies from 0.8 to 5.2/1000 patients in community-based studies while the prevalence ranges between 3.9 and 26.4/1000 live births in hospital-based studies in India, which is not uniform across the country[3,4,67,68]. Ten percent of the present under 5 infant death may be accounted for by CHD.

Malnutrition is a common cause of morbidity in children with congenital heart disease due to frequent hospitalization, poor surgical outcomes, and persistent impairment of somatic growth. The severity of malnutrition ranges from mild under nutrition to failure to thrive. The risk factors for malnutrition in children with congenital heart disease are multifactorial and comprises of poor socioeconomic status, poor IYCF practices, poor parenteral education, heart failure, cyanosis, multiple heart defects, delayed corrective surgery, anemia and pulmonary hypertension.[6]

Anemia was one of the major factor that was reported to aggravate the malnutrition in these children leading to increased risk of mortality and morbidity.

The prevalence of unrecognized anemia in pediatric patients with different congenital heart diseases who were referred for cardiac surgery was found to be rampant.

We conducted a cross sectional study of 80 consenting children aged 1-12 years of age with congenital heart disease chosen by purposive sampling technique, attending as Outpatient and Inpatient in the Department of Pediatrics, Sree Mookambika Institute of Medical Sciences, Kulasekharam over a period of 18 months

AGE DISTRIBUTION:

In the present study, 36.2% belonged to 12mon-59mon and 63.8% belonged to 5-12years.This gives an outlook on the lack of parental education and information regarding their attitude for health care

SEX DISTRIBUTION:

In this study, 40 (50%) were male and 40 (50%) were female children. In a study by Rubia et al,[53] Singh G et al,[69] observed male to female ration 0.9:1 and 1.1:1 respectively and study by Vaidyanathan B et al[41] and Tandaon S et al[70] observed male to female ratio as 1:1 and 0.8:1.

This study shows male to female ratio as 1:1

The bias attitude towards early health care approach and intervention based on gender is changing and equal predilection for both genders is present.

DISTRIBUTION OF SEX ACCORDING TO AGE:

Comparing the distribution of age group and sex, out of the 29 children in the age group 12-59 moths, 48.3% are male and 51.7% are female whereas out of children in the age group 5-12yrs, 51% are female and 49% are male

DISTRIBUTION ACCORDING TO TYPE OF CONGENITAL HEART DISEASE:

Out of the 80 children enrolled with Congenital Heart Disease, majority (81.2%) had acyanotic congenital heart disease and 18.8% had cyanotic congenital heart disease

Majority had VSD-35% followed by ASD 30%, TOF-13.7%,PDA-10% which was again correlating with similar studies by Smitha R et al (VSD-40.7%;ASD-19.06%; PDA-9.53%;TOF13.8%),[4] Kapoor R et al (VSD-21%;ASD-19%;PDA-14.6%;TOF- 4.6%),[71] Mishra et al (VSD-28%;ASD-6%;PDA-8%;TOF-6%),[72] Jatay et al (VSD-28%;ASD-18%;PDA-10%;TOF-6%),[73] Abqari S et al(VSD-38%; ASD-14.75%;PDA-9.5%;TOF13%).[26]

DISTRIBUTION ACCORDING TO SOCIO ECONOMIC STATUS:

In this present study, majority of the children with congenital heart disease belonged to lower (87.4%) class with Upper lower class (66.2%) and lower class (21.2%) according to Modified Kuppusamy Socio Economic Scale. A study by Agha MM et al,[74] Tanden S et al[70] also reported a high prevalence of CHD in children belonging to low socio economic status.

DISTRIBUTION ACCORDING TO FAMILY MEMBERS AFFECTED:

In our study, only one patient had a family member (paternal uncle) who had a history of congenital heart disease (acyanotic) for which surgical correction was done. According to Nelson, the risk of occurrence increases if a 1st degree relative (parent/sibling) is affected (2-6%) whereas when two first degree relatives are affected with CHD, the risk of subsequent child may reach to 20-30%.[2] Oyen N.et al reported that strong familial clustering was present infirst-degree relatives, ranging from 3-fold to 80-fold compared with the population prevalence.[20] Ellesoe GS et al, also reported similar correlation between familial cocurrence and congenital heart disease. [22]

DISTRIBUTION OF CLINICAL FEATURES OF MALNUTRITION:

Clinical features such as clinical pallor, loose skin folds, bipedal edema, vitamin A deficiency, vitamin B complex deficiency, skin changes, hair changes, apathy were considered. In this study, only 25% had pallor had 5% had Vitamin B complex deficiency, 3.8% had Vitamin A and Vitamin D deficiency .None of the children had features of SAM. Okoromah CA et al also concluded from his study that malnutrition in CHD was due to anemia, moderate to severe congestive heart failure (CHF) , poor dietary intake and prolonged unoperated disease.[42] Hassan BA et al also observed that malnutrition correlated significantly with low hemoglobin level, low arterial oxygen saturation, heart failure, pulmonary hypertension, and poor dietary history.[48] Ozkale M et al described in his article that children with malnutrition commonly have anemia which is attributed to bone marrow hypoplasia, iron, vitamin B12, vitamin A and folate deficiency.[11]

DISTRIBUTION ACCORDING TO MATERNAL AGE AT TIME OF CONCEPTION:

In this study, 6.2% of the mothers were below 19 years, 92.5% belonged to 19-35years and 1.2% were above 35 years at the time of conception. Best K.E et al reported that that advanced maternal age is not a risk factor for CHD, however there was marginal risk of infants developing certain types of CHD, among mothers aged more than 35years[27] which was also supported by Miller et al who also observed that infants born to mothers older than 35 years of age seemed to be at 20% increased risk of CHDs, while infants born to younger mothers tended to be at decreased risk of CHDs[23] whereas Luo YL et al observed in his study that the occurrence of CHD was seen in younger mothers.[25]

DISTRIBUTION ACCORDING TO DEGREE OF CONSANGUINITY:

In the study, majority of children with congenital heart disease had parents who were second degree (62.5%) consanguinity and 22.5% were third degree consanguinity and no first degree consanguinity noted. Gnanalingam MG et al also observed that parental consanguinity was noted in 12.5% of the control group compared to 31.1% of the CHD group.[16] A study in Lebanon by Chehab G et al also observed that association between consanguinity mainly first degree cousins, first plus second degree cousins, and any degree of consanguinity, are significantly larger in the occurrence of congenitally malformed hearts.[19]

DISTRIBUTION ACCORDING TO BIRTH ORDER:

In this study, majority of the children with the congenital heart disease belonged to second birth order (56.2%) whereas 31.2% belonged to first and 12.5%

third birth order. A study by Rahman M et al, observed that malnutrition is more rampant with increasing birth order in children in Bangladesh.[75] A similar study by Howell EM concluded that birth order is significantly related to mortality and nutritional status in large African families, with later born children having poorer outcomes.[76]

A possible explanation for this association could be that higher order births are more likely to be unwanted which results in less attention and care from parents: antenatal and postnatal care and child checkup decreases with the higher birth order. Another explanation could be that intra-household allocation of food and resources decreases with an increasing number of births in the household.

DISTRIBUTION ACCORDING TO GESTATIONAL AGE AND BIRTH WEIGHT:

In this study 93.8% were term (>37 weeks) and only 6.2% were preterm (<37weeks) and 82.5% had normal birth weight(2.5-3kg) and only 17.5% out of the total children were low birth weight(<2.5kg). Miller et al reported there was no correlation between the incidence of low birth weight and preterm and congenital heart disease[23] whereas Steurer AM et al, stated that incidence of CCHD was highest at 29 to 31 weeks' GA (0.9%) and lowest at 39 to 42 weeks (0.2%) and that morbidity remains increased across all gestational groups in comparison with infants born at 39 to 42 weeks.[28]

Our study shows that the changing attitude towards periodic antenatal care and care of fetal wellbeing by the mothers as well as the approach by health care workers and health care system in ensuring proper maternal nutrition, antenatal care and safe delivery.

MATERNAL ILLNESS:

None of the mothers had any history of fever, pregnancy induced hypertension, gestational diabetes in our study as these are major risk factors in incidence of congenital heart disease as evidenced by Ramakrishnan A et al,[30] Boyd et al,[31] Tabib et al,[32] Muhammed A et al,[33] Hunter E et al.[34]

DISTRIBUTION ACCORDING TO IYCF PRACTICES:

According to NFHS DATA 4(2016),[77] 54.9% were exclusively breast fed till 6 months, breast feeding was continued in 42.7% up to 2 years, complementary food started at 6 months in 42.7% and family pot feeding was started at 1 year by 14.3% 75 whereas in our study 82.5% were exclusively breast fed till 6 months but only 33.8% were continued breast feeding up to 2 years.

Majority (75%) were started on complementary feeding in the form of mashed rice, mashed idli by end of 6 months and 92.5% were started on family pot feeding by end of 1 year of age.

DISTRIBUTION ACCORDING TO DIETARY INTAKE:

In this study, 5% of the children took <50% of the required calorie and proteins; 93.8% took 50-75% of the required calorie and proteins and only 1.2% took 76-90% of the required calories and protein. Forchielli, M. L.et al in his article described that low energy intake was due to loss of appetite, anoxia, peripheral acidosis, malabsorption.[36]

Jackson M et al also their low energy intakes was due to anoxia, malabsorption and high resting expenditures.[37]

DISTRUBITION ACCORDING TO NUTRITIONAL STATUS (WfA, HfA, WfH, BMI, MUAC):

Studies have suggested that severity of cardiac lesions influence the nutritional status in children with congenital heart disease. Dietary inadequacy, recurrent infections, complications such as heart failure and severe pulmonary hypertension also contribute to malnutrition. As per Baaker et al acute malnutrition(29.5%) is more obvious than chronic malnutrition(21.9%) in patients with CHD and that acute malnutrition is more common in patients with a cyanotic CHD without HF or PH (39.2%), while chronic malnutrition is more obvious in patients with PH and HF (26.3%) and (25%) respectively.[40]

Okoromah CA et al also observed that 90.4% of cases and 21.1% of controls had malnutrition, and 61.2% and 2.6%, respectively, had severe malnutrition. Wasting was significantly higher (58.3%) in acyanotic CHD), and stunting (68.0%) in cyanotic CHD.[42] Oyarzún I. et al reported there was a significant percentage who were underweight (28.3%) and had short stature (21.7%) at the time of admission indicating both acute and chronic malnutrition.[49]Batte and his colleges carried out a similar study in Uganda and observed that 42.5% children were underweight, 45.4% children were stunted, 31.5% were wasted and 27.1% were thin (according to BMI).[47]

In North India, Begum R et al also observed underweight 82.53% in cases vs 24.6% in controls and stunting in 58.72% cases vs 41.26% controls.[53] In South India, Vaidynathan and his colleagues reported prevalence of underweight as 59%, stunting as 26.3% and wasting as 55.9%.[41] Whereas Swagata M et al from

Karnataka observed that 82% were underweight and 86% were stunted among children with congenital heart disease.[50]

Habeeb NM et al, observed that that malnutrition, stunting and wasting were detected in 65.8%, 66.4% and 62.5% of patients respectively and prevalence rates were significantly higher among cyanotics (62.8%, 74.4% and 25.6%) when compared to acyanotics (49.5%, 63.3% and 18.3%)[51] whereas Varan B et al reported mild or borderline malnutrition was more common in group acyanotic CHD with pulmonary hypertension patients whereas both moderate to severe malnutrition and failure to thrive were more common in group cyanotic with pulmonary hypertension patients.[9]

WEIGHT FOR AGE/ WfA

In this present study, according to Weight for Age, 25% were normal and 75% had varying grades of underweight (27.5% had mild, 30% had moderate, 16.2% had severe and 1.2% had very severe underweight), indicating acute malnutrition.

- In children with ACHD, 90.9% had had mild, 70.8% had moderate, 69.2% had severe underweight according to weight for age whereas in children with CCHD, 9.1% had mild, 29.2% had moderate and 35.7% had severe underweight. This was found to be statistically significant with a p value 0.025
- Underweight was more common in children aged 5-12 years (mild- 54.5%, moderate-75%, severe- 71.4%) compared to children between 12-59 months (mild- 4.5%, moderate-25%, severe- 28.5%)

HEIGHT FOR AGE/ HfA

- In this present study, 42.5% were normal and 57.5% had varying grades of stunting (35% had mild, 13.8% had moderate, 8.8% had severe stunting) according to height for age, indicating chronic malnutrition.
- In children with ACHD, 75% had mild, 72.7% had moderate, 42.9% had severe stunting according to height for age whereas in children with CCHD, 25% had mild, 27.3% had moderate, and 57.1% had severe stunting. This was found to be statistically significant with a p value 0.004
- Mild and Moderate stunting was more common in children aged 5-12 years - 64.3% and 63.6% respectively compared to children with 12-59months- 35.7% and 36.4% respectively but severe stunting was common in age group 12-59 months compared to 5-12years (71.4% vs 28.6%)

WEIGHT FOR HEIGHT/ WfH:

- In this study, 16.2% were normal and 83.7% had varying grades of wasting (43.8% had mild, 27.5% had moderate and 12.5% had severe wasting) according to weight for height
- In children with ACHD, 88.6% had mild, 63.6% had moderate, 80% had severe wasting according to weight for height whereas in children with CCHD , 11.4% had mild, 36.4% had moderate, 20% had severe wasting There was no statistical significance
- Wasting was more common in children aged 5-12 years (mild- 68.6%, moderate- 63.6%, severe- 60%) compared to children between 12-59 months (mild- 31.4%, moderate-36.4%, severe- 40%)

CHRONIC ENERGY DENSITY ACCORDING TO BMI:

- In this study, 16.2% were normal, 83.7% had CED. Majority 66.2% had moderate CED and 22.5% had severe CED.
- In children with ACHD, 87.8% had moderate and 55.5% had severe CED according to BMI, whereas in children with CCHD, 12.2% had moderate and 44.5% had severe CED. This was not statistically not significant
- Chronic Energy deficiency based on BMI was more common in children aged 5-12 years (moderate - 61.2 %, severe -7 2.3%) compared to children between 12-59 months (moderate- 38.8%, severe - 27.7%)

MID UPPER ARM CIRCUMFERENCE/MUAC:

- Only 33 children were in the age group of 6-59 months, According to MUAC,51.5% were normal (>13.5cm) and 42.4% were malnourished (27.2% had mild to moderate malnutrition and 15.1% had severe malnutrition)
- .Out of the 5 classified as severe malnutrition, 3 (60%) had <11.5cm classified as Severe acute malnutrition(SAM)

Children with Congenital heart disease had both acute and chronic malnutrition and chronic energy deficiency based on their anthropometric indicators. Children with acyanotic congenital heart disease were more underweight, and wasted and children with cyanotic congenital heart disease were more stunted. Children in age group 5-12 years were more underweight, stunted, wasted compared to children in age group 12-59 months as most of the infants of age group were breast fed and close attention was paid by mothers.

DISTRIBUTION ACCORDING TO DEGREE OF ANEMIA BASED ON HEAMOGLOBIN, RED CELL INDICIES, RDW, PERIPHERAL SMEAR:

Anemia is an important risk factor for morbidity and mortality in patients with cyanotic and acyanotic congenital heart disease .Heart failure may occur and worsen by anemia as a comorbidity. Children with malnutrition commonly have anemia which is attributed to bone marrow hypoplasia, iron, vitamin B12, vitamin A and folate deficiency.[11]

In a study by Gaiha et al, 1993, a prevalence of 18.2% anemia in children with congenital Heart disease was reported.[59] A similar study done in Nairobi by M.O. Lano also reported high prevalence of 16.9% of iron deficiency in children with cyanotic heart disease.[60]

H Amoozgar et al, reported a high prevalence of unrecognized anemia in pediatric patients with different congenital heart diseases who referred for cardiac surgery.[61] A study in Vietnam by Binh TQ et al, reported that 36.1% children in cyanotic group and 24.2% in acyanotic group were diagnosed with true IDA or showed depletion of body iron storage and 77.8% children in cyanotic group and 87.8% in acyanotic group were at risk of iron deficiency.[62]

DISTRIBUTION ACCORDING TO HEAMOGLOBIN:

- In this study, as per hemoglobin estimations and grading anemia according to WHO guidelines, 61.3% of the children with congenital heart disease were normal and 21.3% had anemia (18.8% had mild anemia, 2.5% had moderate anemia). 17.5% had polycythemia

- Even though clinical pallor(Table 12) observed was 25%,the total anemia detected by hemoglobin estimation was only 21.3%
- Hemoglobin is not a sensitive indicator for detecting anemia in CCHD children

DISTRIBUTION ACCORDING TO RED CELL INDICES:

- In this study, 21.2% had all decreased red cell indices and rest(78.8%) had normal red cell indices
- In children with ACHD, 82.5% had normal red cell indices and 76.5% had decreased red cell indices vs children with CCHD, 17.5% had normal red cell indices and 23.5% had decreased red cell indices.

DISTRIBUTION ACCORDING TO RDW:

- In this study 36.2% had increased RDW and 63.8% had normal RDW
- In children with ACHD, 96.1% had normal RDW and only 55.2% had increased RDW whereas children with CCHD, 3.9% had normal RDW and 44.8% had increased RDW. This was statistically significant with p value 0.000.

DISTRIBUTION ACCORDING TO PERIPHERAL SMEAR:

- 16.2% had microcytic hypochromic anemia and the rest(83.8%) had normocytic normochromic anemia
- In children with ACHD, only 76.9% had microcytic hypochromic and rest had normocytic normochromic whereas children with CCHD, 23.1% had microcytic hypochromic and the rest had normocytic normochromic

Hemoglobin, red cell indices were reduced in both ACHD AND CCHD. Even though the children with CCHD did not have clinical pallor or was polycythemic, their red cell indices were greatly reduced red cell indices(23.5%) their RDW was also increased (44.8%) which was statistically significant with p value <0.050. The finding of high prevalence of nutritional anemia especially iron deficiency anemia shows the effect of poor diet and also paves the path for its corrective interventions like iron supplementation in optimum doses for prevention (2-3mg/kg/day) or treatment(4-6mg/kg/day).

Introduction

Aims & Objectives

Review of Literature

Materials & Methods

Results

Scientific Justification & Hypothesis

Discussion

Summary

Limitations

Conclusion

Bibliography

Appendix

Abstract

SUMMARY

Among the 80 children with congenital heart disease, in the present study, 36.2% belonged to 12 month - 59 month and 63.8% belonged to 5-12 years. This gives an outlook on the lack of parental education and information regarding their attitude for health care.

81.2% had acyanotic congenital heart disease (VSD-43.1%, ASD-36.9%, 12.3% PDA) and 18.8% had cyanotic congenital heart disease (TOF-73.3%).

Majority of the children with congenital heart disease belonged to lower (87.4%) class with Upper lower class (66.2%) and lower class (21.2%) according to Modified Kuppusamy Socio Economic Scale.

In this study, only 25% had clinical pallor had 5% had Vitamin B complex deficiency, 3.8% had Vitamin A and Vitamin D deficiency .None of the children had features of SAM. None of the children had features of SAM

In this study, 6.2% of the mothers were below 19 years, 92.5% belonged to 19-35years and 1.2% were above 35 years at the time of conception.

Majority of children with congenital heart disease had parents who were second degree (62.5%) consanguinity and 22.5% were third degree consanguinity but no first degree consanguinity. Majority of the children with the congenital heart disease belonged to second birth order (56.2%) whereas 31.2% belonged to first and 12.5% third birth order.

In this study 93.8% were term (>37 weeks) and only 6.2% were preterm (<37weeks) and 82.5% had normal birth weight (2.5-3kg) and only 17.5% out of the total children were low birth weight(<2.5kg) .

82.5% were exclusively breast fed till 6 months but only 33.8% were continued breast feeding upto 2 years. Majority (75%) were started on complementary feeding in the form of mashed rice, mashed idli by end of 6 months and 92.5% were started on family pot feeding by end of 1 year of age.

Majority (93.8%) took 50-75% of the required calorie and proteins according to ICMR 2010

According to Weight for Age, 25% were normal and 75% had varying grades of underweight (27.5% had mild, 30% had moderate, 16.2% had severe and 1.2% had very severe underweight), indicating acute malnutrition. There was significant underweight in children with ACHD underweight compared to CCHD with a p value 0.025

According to Height for Age, 42.5% were normal and 57.5% had varying grades of stunting (35% had mild, 13.8% had moderate, 8.8% had severe stunting) age, indicating chronic malnutrition. Severe stunting was significant in children with CCHD compared to ACHD with a p value 0.004

According to Weight for Height, 16.2% were normal and 83.7% had varying grades of wasting (43.8% had mild, 27.5% had moderate and 12.5% had severe wasting) Wasting was more predominant in children with ACHD compared to children with CCHD. There was no statistical significance.

In this study group, as per BMI, 16.2% were normal, 83.7% had chronic energy deficiency (CED). In ACHD children, CED was more predominant vs children with CCHD.

Children in age group 5-12 years were more underweight, stunted, wasted compared to children in age group 12-59 months as most of the infants of age group 12 months - 59 months were breast fed and close attention was paid by mothers.

Only 33 children were in the age group of 6-59 months, according to MUAC, 51.5% were normal (>13.5cm) and 42.4% were malnourished (27.2% had mild to moderate malnutrition and 15.1% had severe malnutrition). Out of the 5 classified as severe malnutrition, 3 had <11.5cm classified as Severe Acute Malnutrition (SAM).

In this study, as per hemoglobin estimations and grading anemia according to WHO guidelines, 61.3% of the children with congenital heart disease were normal and 21.3% had anemia .17.5% had polycythemia. Hemoglobin is not a sensitive indicator in detecting anemia in CCHD children

In this study, 21.2% had all decreased red cell indices (MCV, MCH, MCH) and rest (78.8%) had normal red cell indices. Red Cell Indices were reduced in both ACHD and CCHD (76.5% vs 23.5%)

In the present study, 36.2% had increased RDW and 63.8% had normal RDW.RDW was increased more in children with ACHD compared to CCHD (55.2% vs44.8%). This was statistically significant with p value 0.000.

In this study, 16.2% had microcytic hypochromic anemia and the rest (83.8%) had normocytic normochromic anemia according to peripheral smear.

As evidenced by peripheral smear, Microcytic hypochromic anema was more common in in children with ACHD, compared to CCHD (76.9% vs 23.1%).

LIMITATIONS OF THE STUDY

1. The distribution of age group and the type of CHD in the study population was non uniform
2. The nutritional and definitive interventions were not assessed or followed up as this was a cross sectional study

CONCLUSION

Congenital heart defect (CHD) is the most common congenital malformation among all birth defects leading to morbidity and mortality among children. The burden of CHD is high in developing countries like India, due to the high birth rate and critical nature of CHD requiring expensive surgical and non-surgical interventions. Malnutrition and anemia is rampant among children with CHD with a significant impact on the intervention and the outcome of intervention.

In this study on 80 children, 63.8 % were in the age group 1-12 years whereas 36.2% belonged to age group 12mon-59mon.The male to female ratio was 1:1. 81.3% had ACHD; out of which VSD was the most common (35%).18.7% had CCHD, out of which TOF was the most common (13.7%).

69.2% of ACHD were underweight in comparison to 35.7% in CCHD.,42.9% of ACHD were stunted in comparison to 57.1% in CCHD,80% of ACHD were wasted in comparison to 20% wasted in CCHD.

21.3% of ACHD had anemia 17.5% of CCHD had polycythemia.21.2% had decreased red cell indices indicating microcytic hypochromic and 36.2% had increased RDW indicating nutritional anemia. According to Peripheral smear, 16.2% had microcytic hypochromic anemia.

The high proportion of malnutrition and anemia among children with CHD warrants proper evaluation and early intervention. This is of utmost importance as majority of CHD are likely to get surgical and non-surgical intervention under the RBSK scheme. The RBSK scheme also focuses on malnutrition and deficiency disorder.

Accreditation of private institution under the RBSK scheme for intervention of CHD is a big boon to the community.

BIBLIOGRAPHY

1. Dolk H, Loane M, Garne E. Congenital heart defects in Europe: Prevalence and perinatal mortality, 2000 to 2005. Circulation 2011;123:841-9.
2. Bernstein D, Epidemiology and genetic basis of congenital heart disease. In: Behrman RE(ed). Nelson Textbook of Pediatrics.20th edition, Philadelphia. Elsevier; 2016.p.2182-86
3. Bhat NK, Dhar M, Kumar R, Patel A, Rawat A, Kalra BP. Prevalence and pattern of congenital heart disease in Uttakhand, India. Indian J Pediatr.2013;80:281-5.
4. Smitha R, Karat SC, Narayanappa D, Krishnamurthy B, Prasanth SN, Ramachandra B, et al. Prevalence of congenital heart diseases in Mysore. Indian J Hum Genet. 2006; 12:11-6.
5. Kumar K, Complications of Congenital Heart Disease. In: Parthasarathy A, Gupta P(ed). IAP Textbook of Pediatrics. 6th edition. New Delhi. Jaypee Pub; 2016. p.497-99
6. Strangway A, Fowler R, Cunningham K, Hamilton JR. Diet and growth in congenital heart disease. Am J Paediatrics 1976; 57:75-86.
7. Varan B, Tokel K, Yilmaz G. Malnutrition and growth failure in cyanotic and a cyanotic congenital heart disease with and without pulmonary hypertension. Arch Dis Child.1999;81:49-52.
8. Krieger I. Growth failure and congenital heart disease. Energy and nitrogen balance in infants. Am J Dis Child 1970;120:497-502.

9. Pittman JG, Cohen P. The pathogenesis of cardiac cachexia. N Engl J Med 1964; 271:403-9.

10. Rodica T. Nutritional Approach of Pediatric Patients Diagnosed with Congenital Heart Disease. Pediatric Cardiology Clinic 2013;59(2):121-5.

11. Ozkale M, Sipahi T. Hematologic and bone marrow changes in children with protein-energy malnutrition. Pediatr Hematol Oncol 2014;31(4):349-58.

12. Carson JA, Duff A, Poses RM, Berlin JA, Spence RK, Trout R, et al. Effect of anemia and cardiovascular disease on surgical mortality and morbidity. Lancet 1996; 348:1055-60

13. Bernstein D. Cardiac Development. In: Behrman RE(ed). Nelson Textbook of Pediatrics.20th edition, Philadelphia. Elsevier; 2016:p. 2157-59

14. Abdulla R, Blew GA, Holterman MJ. Cardiovascular Embryology. Pediatr Cardiol 2004; 25:191-200.

15. Bernstein D. Fetal to Neonatal Circulatory Transition. In: Behrman RE(ed). Nelson Textbook of Pediatrics 20th edition. Philadelphia. Elsevier; 2016:p.2161-2

16. Gnanalingam MG, Gnanalingam KK, Singh A. Congenital heart disease and parental consanguinity in South India. Acta Paediatr 1999;88:473-4

17. Becker SM, Al Halees Z, Molina C, Paterson RM. Consanguinity and congenital heart disease in Saudi Arabia. Am J Med Genet 2001;99:8-13.

18. Yunis K, Mumtaz G, Bitar F, Chamseddine F, Kassar M, Rashkidi J. Consanguineous marriage and congenital heart defects: a case-control study in the neonatal period. Am J Med Genet Part A 2006;140:1524-30.

19. Chehab G, Chedid P, Saliba Z, Bouvagnet P. Congenital cardiac disease and inbreeding: specific defects escape higher risk due to parental consanguinity. Cardiol Young 2007;17:414-22.
20. Oyen N, Poulsen G, Boyd HA, Wohlfahrt J, Jensen PKA,Melbye M.Recurrence of Congenital Heart Defects in Families. Circulation 2009;20(4):295-301.
21. Peyvandi S, Ingall E, Woyciechowski S. Risk of congenital heart disease in relatives of probands with conotruncal cardiac defects: An evaluation of 1,620 families. Am J Med Genet Part A 2014; 9999:1-6.
22. Ellesoe GS, Workman TC, Bouvagnet P. Familial co-occurrence of congenital heart defects follows distinct patterns. European Heart Journal 2018; 39(12):1015-22
23. Miller A, Riehle CT, Siffel C, Frias JL, Correa A. Maternal age and prevalence of isolated congenital heart defects in an urban area of the United States. Am J Med Genet Part A 2011; 9999:1-9.
24. Gill SK, Broussard C, Devine O. Association between Maternal Age and Birth Defects of Unknown Etiology, 1997-2007. Clinica Mole Terat 2012; 94(12):1010-18.
25. Luo YL, Cheng YL, Gao XH, Tan SQ, Li JM, Wang W.Maternal Age, Parity and Isolated Birth Defects: A Population-Based Case-Control Study in Shenzhen. China 2013;8(11): e81369
26. Abqari S, Gupta A, Shahab T, Rabbani MU, Ali SM, Firdaus U. Profile and risk factors for congenital heart defects: A study in a tertiary care hospital. Ann Pediatr Card 2016;9:216-21.

27. Best KE, Rankin J. Is advanced maternal age a risk factor for congenital heart disease?. Clinic Mole Terat 2016;106(6);461-7.
28. Steurer MA, Baer RJ, Keller RL. Gestational Age and Outcomes in Critical Congenital Heart Disease. Pediatrics 2017;140(4):e20170999
29. Auger N, Fraser WD, Profitos JH, Arbour L. Association between Preeclampsia and Congenital Heart Defects. J Am Med Assc 2015;314(15):1588-98.
30. Ramakrishnan A, Lee LJ, Mitchell LE, Agopian AJ. Maternal Hypertension During Pregnancy and the Risk of Congenital Heart Defects in Offspring: A Systematic Review and Meta-analysis. Pediatr Cardiol 2015;36(7): 1442-51.
31. Boyd HA, Basit S, Behrens I,Leirgul E, Bundgaard H. Association Between Fetal Congenital Heart Defects and Maternal Risk of Hypertensive Disorders of Pregnancy in the Same Pregnancy and Across Pregnancies. Am Heart Assc Circulation 2017;136:39-48.
32. Tabib A, Shirzad N, Sheikhbahaei S. Cardiac Malformations in Foetuses of Gestational and Pre Gestational Diabetic Mothers. Iranian J Pediatr 2013; 23(6):664-8.
33. Muhammad A, Khan M, Khan I, Anwar T. Frequency of congenital heart diseases in infants of diabetic mothers referred to pediatrics department. J Postgrad Med Inst 2014; 28(1):37-41.
34. Hunter LE, Sharland GK. Maternal Gestational Diabetes and fetal Congenital Heart Disease: An Observational Study. J Preg Child Health 2015;2:132.
35. Arjmandnia M, Besharati M, Rezvan S. Studying the determinant factors leading to congenital heart disease in newborns. J Edu Health Promot 2018;7:53-6.

36. Forchielli ML, McColl R, Walker WA, Lo C. Children with Congenital Heart Disease: A Nutrition Challenge. Nutrition Reviews 2009;52(10):348-53.
37. Jackson M, Poskitt EM. The effects of high-energy feeding on energy balance and growth in infants with congenital heart disease and failure to thrive.Br J Nutr 1991;65(2):131-43.
38. Hubschman LE.Malnutrition in Congenital Heart Disease, Management to Improve Outcome. SAGE Jr 2013;5(3):170-76.
39. Mitchell M, Logan RW, Pollock JCS, Jamieson MPG. Nutritional status of children with congenital heart disease. Br Heart Jr 1995;73:277-83.
40. Baaker RH, Abass AA, Kamel AA. Malnutrition and Growth Status in Patients with Congenital Heart Disease. Iraqi PG Med Jr 2008;7(2):152-5.
41. Vaidyanathan B, Nair SB, Sundaram KR, Babu UK, Shivaprakaste K, Rao SG.Malnutrition in children with congenital heart disease (CHD): determinants and short-term impact of corrective intervention. Indian Pediatr 2008; 45: 541-6.
42. Okoromah CA, Ekure EN, Lesi FE, Okunowo WO, Tijani BO, Okeiyi JC. Prevalence, profile and predictors of malnutrition in children with congenital heart defects: a case-control observational study 2011;96(4):354-60.
43. Vaidyanathan B, Radhakrishnan R, Sarala DA, Sundaram KR, Kumar RK. What determines nutritional recovery in malnourished children after correction of congenital heart defects? Indian Pediatr 2009;124(2):294-9.
44. Ratanachu S, Pongdara A. Nutritional Status of Pediatric Patients with Congenital Heart Disease: Pre- and Post Cardiac Surgery. J Med Assoc Thai 2011;94(3): 133-7.

45. Daymont C, Neal A, Prosnitz A, Cohen MS. Growth in Children With Congenital Heart Disease Pediatrics 2013;131(1):236-242.
46. Arodiwe I, Chinawa J, Ukoha M, Ujunwa F, Adiele D, Obidike E. Nutritional status of congenital heart disease (CHD) patients:Burden and determinant of malnutrition at university of Nigeria teaching hospital Ituku – Ozalla, Enugu. Pak J Med Sci 2015;31(5):1140-45.
47. Batte A, Lwabi P, Lubega S, Kiguli S. Wasting, underweight and stunting among children with congenital heart disease presenting at Mulago hospital, Uganda. BMC Pediatric.2017;17(10):1-7
48. Hassan BA, Albanna EA, Morsy SM, Siam AG, Al Shafie MM, Elsaadany HF, et al. Nutritional Status in Children with Un-Operated Congenital Heart Disease: An Egyptian Center Experience. Front Pediatr 2015;3:53.
49. Oyarzun I, Claveria C, Larios G, Le Roy C. Nutritional recovery after cardiac surgery in children with congenital heart disease. Rev Chil Pediatr 2018;89(1):24-31
50. Swagata M, D'Souza J, Anthropometric profiles of children with congenital heart disease. Int J Pediatr Res 2016;3(8):577-583
51. Habeeb NM, Al-Fahham MM, Tawfik, Mohammed MM. Nutritional Assessment of Children with Congenital Heart Disease - A Comparative Study in Relation to Type, Operative Intervention and Complications. EC Paediatrics 2017;6(4): 112-120.
52. Noori NM, Moghaddam NM, Teimouri A, Boryri T, Abadi HS. Evaluation of Growth Status in Children with Congenital Heart Disease: A Case- Control Study. Int J Pediatr 2017;5(12):6503-14.

53. Begum R, Kher A. Anthropometric assessment in children with congenital heart disease Int J Contemp Pediatr.2018;5(2):634-639

54. Bukovac LT. Growth and development in children with congenital heart defects. Lijec Vjesn 1993;115:79-84.

55. Leite HP, Fisberg M. Nutritional status of children with congenital heart disease and left-to-right shunt. The importance of the presence of pulmonary hypertension. Arq Bras Cardiol 1995;65(5):403-7.

56. Blasquez A, Clouzeau H, Fayon M, Mouton JB, Thambo JB, Enaud R, Lamireau T. Evaluation of nutritional status and support in children with congenital heart disease. Eur J Clin Nutr 2016;70:528-31.

57. El-Koofy N, Mahmoud AM, Fattouh AM, Nutritional rehabilitation for children with congenital heart disease with left to right shunt. The Turkish Journal of Pediatrics 2017; 59:442-451

58. Balogun FM, Omokhodion SI. Nutritional profiles and selected parental factors among children with congenital heart diseases in Ibadan, Nigeria. Nig J Cardiol 2015;12:89-94

59. Gaiha M, Sethi H, Sudha R. Clinico-hematological study of iron deficiency anemia and its correlation with hyper viscosity symptoms in cyanotic congenital heart disease. Indian Heart J 1993;45:53-55.

60. Lang'o M, Githang's Yuko-Jowi CA. Prevalence of iron deficiency in children with cyanotic congenital heart disease seen at Kenyatta National Hospital. East Afr Med J 2009;86:47-51

61. Amoozgar H, Soltani M, Besharati A, Cheriki S. Undiagnosed Anemia in Pediatric Patients with Congenital Heart Diseases. Int Cardiovas Res J 2011; 5(2):70-71.

62. Binh TQ, Suong NTB, Dinh NH. Iron deficiency anemia in children with congenital heart disease and the usefulness of erythrocyte indexes as a screening tool. Arch Gen Intern Med 2018;2(3):1-4.

63. Elizabeth K.E, Pedibloom:Pediatric Cases and Summaries. New Delhi. Jaypee Pub India;2012:p.27-9

64. WHO. Haemoglobin concentrations for the diagnosis of anaemia and assessment of severity. Vitamin and Mineral Nutrition Information System. Geneva, World Health Organization, 2011 (WHO/NMH/NHD/MNM/11.1) (http://www.who.int/ vmnis/ indicators/haemoglobin. pdf, accessed [date])

65. Tefferi A,Vardiman JW.Classification and diagnosis of myeloproliferative neoplasms: the 2008 World Health Organization criteria and point of care diagnostic algorithms. Leukemia 2008;22:14-22

66. Lo SF. Reference Intervels for laboratory tests and procedures In: Behrman RE(ed). Nelson Textbook of Pediatrics. 20th edition. Philadelphia;2016:p.3466

67. Vashishtha VM, Kalra A, Kalra K, Jain VK. Prevalence of congenital heart disease in school children. Indian Pediatr 1993;30:1337-40

68. Saxena A. Congenital heart disease in India: A status report. Indian J Pediatr 2005;72:595

69. Singh D, Singh G. Gender equality in India for children with congenital heart disease: Looking for answers. Brit Med J 2011;97:290-98.

70. Tandon A, Sengupta S, Shukla V. Risk factors for congenital heart disease in Vellore. Curr Res J Biol Sci 2010;2(4):253-8.

71. Kapoor R, Gupta S. Prevalence of congenital heart disease, Kanpur, India. Indian Pediatr 2008;45:309-11.

72. Mishra M, Mittal M, Verma AM, Rai R, Chandra G, Singh DP. Prevalence and pattern of congenital heart disease in school children of Eastern Uttar Pradesh. Indian Heart J 2009;61:58-60.

73. Jatav RK, Kumbhare MB, Srinivas M, Rao DR, Kumar PG, Reddy PR. Prevalence and pattern of congenital heart diseases in Karimnagar, Andhra Pradesh, India:diagnosed clinically and by trans-thoracic-two dimensional echocardiography. Int J Res Med Sci 2014;2:186-92.

74. Agha MM, Glazier RH, Moineddin R. Socioeconomic status and prevalence of congenital heart disease: Does universal access to health care system eliminate the gap. Birth Defects Res A Clin Mol Teratol 2011;91(12):1011-8.

75. Rahman M. Association between order of birth and chronic malnutrition of children. Cad Saude Publica 2016;32(2):e00011215

76. Howell ME,Holla N, Waidmann T.Being the younger child in a large African Family: a study of birth order as a risk factor for poor health using the demographic and health surveys for 18 countries. Bio Med Control Nutri 2016;2(61):1-12

77. National Family Health Survey-4 2015-16. India Fact sheets. rchiips.org/NFHS/fact sheet NFHS-4, Available at: http://www.mohfw.nic.in Date accessed: 03 Sep. 2017.

APPENDIX - 1

SREE MOOKAMBIKA INSTITUTE OF MEDICAL SCIENCES

KULASEKHARAM

RESEARCH COMMITTEE

CERTIFICATE

This is to certify that The Research Protocol Submitted by Dr. NARGIS RABIYA. A.N

Faculty / Post Graduate from Department of PAEDIATRICS

Titled ASSESSMENT of NUTRITIONAL STATUS AND ANEMIA IN CHILDREN WITH CONGENITAL HEART DISEASE

is approved by the Research Committee.

Chair Person

Prof. & H.O.D.
Dept. of Bio-Chemistry
Sree Mookambika Institute of Medical Sciences
Kulasekharam 629 161

Convenor

Prof. & H.O.D.
Dept. of Physiology
Sree Mookambika Institute of Medical Sciences
Kulasekharam 629 161

Date :

APPENDIX - 2

INSTITUTIONAL HUMAN ETHICS COMMITTEE

SREE MOOKAMBIKA INSTITUTE OF MEDICAL SCIENCES,
KULASEKHARAM, TAMILNADU

Communication of Decision of the Institutional Human Ethics Committee(IHEC)

SMIMS/IHEC No: 1 /Protocol no:28 / 2016

Protocol title: ASSESSMENT OF NUTRITIONAL STATUS AND ANEMIA IN CHILDREN WITH CONGENITAL HEART DISEASE
Principal Investigator: Dr. Nargis Rabiya .A.N
Name& Address of Institution: Department of Paediatrics Sree Mookambika Institute of Medical Sciences, Kulasekharam ☑ New review ☐ Revised review ☐ Expedited review
Date of review (D/M/Y): 15.12.2016 Date of previous review , if revised application:
Decision of the IHEC: ☑ Recommended ☐ Recommended with suggestions ☐ Revision ☐ Rejected
Suggestions/ Reasons/ Remarks:
Recommended for a period of : eighteen months

Please note*

- Inform IHEC immediately in case of any Adverse events and Serious adverse events.
- Inform IHEC in case of any change of study procedure, site and investigator
- This permission is only for period mentioned above. Annual report to be submitted to IHEC.
- Members of IHEC have right to monitor the trial with prior intimation

Reneegalgangadhar
Signature of Member/Secretary IHEC

SREE MOOKAMBIKA INSTITUTE OF MEDICAL SCIENCES ★ KULASEKHARAM ★
IHEC
Date 21/12/16

APPENDIX - 3

CASE RECORD FORM

Serial no	**:**			
Date	**:**			
Age in years:				
Address & Phone no	**:**	.....		
Sex	**:**	**1. Male**	**2. Female**	
Socioeconomic Status				
Kuppuswami Scale	**:**	I II III IV V →**1/2/3/4/5**		
Diagnosis	**:**	**ACHD/CCHD (A/C)**		
Comorbidity	**:**	**YES/NO (1/0)**		
Anemia	**:**	No pallor/Some Pallor/Severe Pallor **0/1/2**		
Clinical Features of Malnutrition	**:**	Loose skin folds	YES/NO	(1/0)
		Bipedal Edema	YES/NO	(1/0)
		Vitamin A Deficiency	YES/NO	(1/0)
		B complex deficiency	YES/NO	(1/0)
		Vitamin D Deficiency	YES/NO	(1/0)
		Skin changes	YES/NO	(1/0)
		Hair changes	YES/NO	(1/0)
		Apathy	YES/NO	(1/0)
ECHO	**:**			
Other Affected Members	**:**	**YES(1)/NO(0)/SIBLING 1A/PARENT 1B**		
Maternal Characteristics	**:**			
Age of Conception	**:**			
Birth Order	**:**			
Obstetric/Medical	**:**	**NONE(0) / PIH(1) / GDM(2) / OTHERS(3)**		
CHILD				
Breast Feeding	**:**	Exclusive up to 6 months **YES/NO (1/0)**		
		Continued up to 2 years **YES/NO (1/0)**		

Complementary Feeding : Semisolids at 6 months **YES/NO (1/0)**

Family Pot Feeding : Started by 1 year **YES/NO (1/0)**

Dietary adequacy :

- Energy : **<50% / 50-75% / 76-90% / >90% (1/2/3/4)**
- Protein : **<50% / 50-75% / 76-90% / >90%(1/2/3/4)**
- Micronutrients : **YES/NO (1/0)**

Birth Details : **TERM/PT(1/2) NBW/LBW(1/2)**
(NBW (Normal Birth Weight)>=2500gms/LBW (Low birth weight) <2500gms / PT (Preterm) <37 weeks / Term >=37weeks)

ANTHROPOMETRIC ASSESSMENT:

1. **WEIGHT FOR AGE: Normal/Mild underweight (grade I)/Moderate underweight (grade II)/ Severe underweight (grade III)/Very severe underweight (grade IV) [0/1/2/3/4/5]**
2. **HEIGHT FOR AGE : Normal/Mild stunting (I degree)/ Moderate stunting (II degree)/ Severe stunting (III degree) [0/1/2/3]**
3. **WEIGHT FOR HEIGHT: Normal/Mild wasting/(I degree)/Moderate wasting(II degree)/Severe wasting(III degree)[0/1/2/3]**
4. **BMI : < 5yrs: <3rd centile/3rd-50th centile/50th-85th centile/ 85th-95th centile/>95th centile[1/2/3/4/5]**
   >5yrs: <3rd centile/3rd-50th centile/50th -23rd equivalent/ 23rd-27th equivalent/>27th equivalent[1/2/3/4/5]
5. **MUAC : Normal(>13.5)/Mild-Moderate(12.5 13.5)/Severe(<12.5)/SAM(<11.5)[0/1/2/3]**

Investigations

Hb (Anemia) :

Peripheral Smear : Microcytic Hypochromic/Megaloblastic Normochromic/Normocytic Normochromic **(1/2/3)**

APPENDIX – 4

MASTER CHART

S.NO	AGE	SEX	SES	DIAG	TYPE	COMORB		CFLSKIN	CFEDEMA	CFVItA	CFBCOM	CFVITD	CFSKIN	CFHAIR	CFAPATH	OTHMEM	MAGE	CONS	BO	OBGILL	EBF6MO	BF2YR
1	4 yrs	1	4	1	1	0	0	0	0	0	1	0	0	0	0	0	24	1	2	0	0	0
2	1YR 6 MON	2	4	1	4	0	0	0	0	0	1	0	0	0	0	0	23	1	2	0	1	1
3	1YR 7MON	1	4	1	4	0	0	0	0	1	1	0	0	0	0	0	27	1	2	0	1	1
4	12 YRS	1	4	1	3	0	0	0	0	0	1	0	0	0	0	0	24	1	2	0	1	0
5	7 YRS	2	5	1	1	0	0	0	0	0	0	0	0	0	0	0	26	1	3	0	1	1
6	4 YRS	2	4	2	1	0	0	0	0	0	0	1	0	1	0	0	25	0	2	0	1	1
7	5 YRS	2	5	2	1	0	0	0	0	0	0	0	0	0	0	0	17	1	2	0	1	1
8	10 YRS	2	5	1	3	0	0	0	0	1	0	0	0	0	0	0	22	1	2	0	0	0
9	11YR 2 MON	2	4	1	1	0	0	0	0	0	0	0	0	0	0	0	21	1	2	0	0	0
10	6 YRS	1	5	1	1	0	0	0	0	0	0	0	0	0	0	1	24	1	2	0	1	1
11	11YRS	1	5	1	1	0	0	0	0	0	0	1	0	0	0	0	27	1	2	0	1	1
12	7YRS	2	5	1	1	0	0	0	0	0	0	0	0	1	0	0	30	1	2	0	1	1
13	4YRS 8 MONS	2	5	2	1	0	0	0	0	1	0	0	0	0	0	0	23	1	2	0	1	1
14	3YRS	2	5	2	1	0	0	0	0	0	0	0	0	1	0	0	27	1	2	0	1	0
15	9YR 3 MONS	2	5	1	2	0	0	0	0	0	0	0	0	1	0	0	23	1	2	0	1	0
16	7YRS	2	4	1	1	0	0	0	0	0	0	0	0	0	0	0	23	0	1	0	0	0
17	12YRS	1	5	1	3	0	0	0	0	0	0	0	0	0	0	0	32	1	2	0	1	0
18	3YRS 9 MONS	2	5	1	1	0	0	0	0	0	0	0	0	0	0	0	25	1	3	0	1	1

19	9YRS	2	4	1	2	0	0	0	0	0	0	0	0	0	0	0	23	1	2	0	1	0
20	7 YRS	2	4	1	1	0	0	0	0	0	0	0	0	0	0	0	23	1	2	0	1	0
21	9 YRS	1	4	1	3	0	0	0	0	0	0	0	0	0	0	0	21	1	1	0	0	0
22	8YRS	1	3	1	2	0	0	0	0	0	0	0	0	0	0	0	26	1	1	0	0	0
23	9 YRS	1	3	1	2	0	0	0	0	0	0	0	0	0	0	0	23	1	2	0	0	0
24	10 YRS	2	2	1	1	0	0	0	0	0	0	0	0	1	0	0	28	1	3	0	1	0
25	4 YRS	1	4	1	1	0	0	0	0	0	0	0	0	0	0	0	23	1	3	0	1	1
26	9 YRS	1	4	2	1	0	0	0	0	0	0	1	0	0	0	0	22	1	2	0	1	0
27	5YRS	1	4	2	2	0	0	0	0	0	0	0	1	0	0	0	21	0	1	0	1	1
28	8 YRS	1	4	1	1	0	0	0	0	0	0	0	1	0	0	0	24	0	2	0	0	0
29	7YR 2 MON	1	4	1	1	0	0	0	0	0	0	0	0	0	0	0	21	0	1	0	1	0
30	6 YRS	2	4	1	1	0	0	0	0	0	0	0	0	0	0	0	21	1	1	0	1	0
31	12YRS	2	4	1	1	0	0	0	0	0	0	0	0	0	0	0	23	1	1	0	1	1
32	9YRS	2	5	1	4	0	0	0	0	0	0	0	0	0	0	0	26	1	2	0	1	0
33	9YRS	2	4	1	2	0	0	0	0	0	0	0	0	1	0	0	19	1	2	0	1	0
34	9YRS 8 MONS	2	3	1	4	0	0	0	0	0	0	0	1	0	0	0	41	1	3	0	1	1
35	10 YRS	1	4	1	2	0	0	0	0	0	0	0	0	0	0	0	23	1	2	0	1	0
36	11 YRS	2	4	1	1	0	0	0	0	0	0	0	0	0	0	0	30	1	2	0	1	1
37	12 YRS	1	4	1	2	0	0	0	0	0	0	0	0	0	0	0	19	1	1	0	1	0
38	7 YRS	1	5	1	1	0	0	0	0	0	0	0	0	0	0	0	20	1	1	0	1	0
39	8 YRS	1	5	1	2	0	0	0	0	0	0	0	0	0	0	0	27	0	2	0	0	0
40	9 YRS	1	4	1	2	0	0	0	0	0	0	0	0	0	0	0	21	1	1	0	1	0
41	9 YRS	1	4	2	1	0	0	0	0	0	0	0	1	0	0	0	26	1	3	0	1	0
42	9 YRS	1	4	1	2	0	0	0	0	0	0	0	0	0	0	0	18	1	1	0	1	0
43	8YRS	2	4	1	2	0	0	0	0	0	0	0	0	0	0	0	24	1	2	0	1	0

44	7 YRS	2	4	2	2	0	0	0	0	0	0	0	0	0	0	0	22	0	1	0	1	1
45	7YRS 7 MON	1	4	1	1	1	0	0	0	0	0	0	0	0	0	0	23	0	2	0	1	0
46	10 YRS	1	4	1	2	0	0	0	0	0	0	0	0	0	0	0	22	1	1	0	0	0
47	7YRS	1	4	1	2	0	0	0	0	0	0	0	0	0	0	0	24	1	2	0	1	0
48	8YRS	2	4	2	2	0	0	0	0	0	0	0	0	0	0	0	17	1	2	0	0	0
49	12YRS	2	4	1	2	0	0	0	0	0	0	0	0	0	0	0	26	1	1	0	1	0
50	6YRS 11 MONTS	1	4	1	1	0	0	0	0	0	0	0	0	0	0	0	21	1	2	0	1	0
51	5YRS 6 MONS	1	4	1	1	0	0	0	0	0	0	0	0	0	0	0	26	0	2	0	1	0
52	10YRS	1	4	1	2	0	0	0	0	0	0	0	0	0	0	0	20	0	3	0	1	0
54	12YRS	1	4	1	4	0	0	0	0	0	0	0	0	0	0	0	23	1	2	0	1	0
55	1YRS 6 MONS	1	4	1	3	1	0	0	0	0	0	0	0	0	0	0	23	1	1	0	1	0
56	9 YRS	1	4	1	2	0	0	0	0	0	0	0	0	1	0	0	23	1	3	0	1	0
57	3 YRS	2	5	1	2	0	0	0	0	0	0	0	0	0	0	0	20	1	2	0	1	0
58	2YRS	2	3	2	1	0	0	0	0	0	0	0	0	1	0	0	29	0	1	0	1	0
59	4YRS	1	3	1	1	0	0	0	0	0	0	0	0	0	0	0	23	1	1	0	1	1
60	3 YRS 6 MONS	1	2	1	2	0	0	0	0	0	0	0	1	0	0	0	30	1	2	0	1	0
61	1YRS 6 MONS	1	4	1	2	0	0	0	0	0	0	0	1	0	0	0	23	1	2	0	1	0
62	1 YR	2	3	1	2	0	1	0	0	0	0	0	0	0	0	0	22	1	2	0	1	0
63	4 YRS	1	4	1	1	0	1	0	0	0	0	0	0	0	0	0	29	1	2	0	1	0
64	6 YRS	2	4	2	1	0	1	0	0	0	0	0	0	0	0	0	20	1	1	0	1	1
65	1YR 1 MON	1	4	1	3	0	1	0	0	0	0	0	0	1	0	0	26	1	1	0	1	1
66	1YR 6 MON	2	5	2	1	0	1	0	0	0	0	0	0	0	0	0	21	1	2	0	1	0
67	3 YR	1	2	1	2	0	1	0	0	0	0	0	0	0	0	0	30	1	2	0	1	1
68	4 YR 7 MON	2	4	1	1	0	1	0	0	0	0	0	0	0	0	0	30	1	2	0	1	1
69	2 YR	2	4	1	3	0	1	0	0	0	0	0	0	0	0	0	24	1	1	0	1	1

70	1YR 7MON	2	4	1	3	0	1	0	0	0	0	0	0	0	0	0	23	1	2	0	1	1
71	3YR	1	4	1	2	1	1	0	0	0	0	0	0	0	0	0	22	1	1	0	1	0
72	1YR 6 MON	2	4	1	2	0	1	0	0	0	0	0	0	0	0	0	30	1	2	0	0	0
73	1YR 3 MON	1	5	1	1	1	1	0	0	0	0	0	0	0	0	0	28	1	1	0	0	0
74	5YR	2	4	2	1	0	1	0	0	0	0	0	0	0	0	0	24	1	1	1	1	1
75	3YR	2	4	1	2	0	1	0	0	0	0	0	0	0	0	0	23	1	2	0	1	1
76	1 YR	1	4	2	1	0	1	0	0	0	0	0	0	0	0	0	29	1	3	0	1	1
77	3YR	1	4	2	2	0	1	0	0	0	0	0	0	0	0	0	28	1	3	0	1	0
78	10 YRS	2	3	1	2	0	2	0	0	0	0	0	0	0	0	0	28	1	2	0	1	0
79	11 YRS	2	4	1	2	0	2	0	0	0	0	0	0	1	0	0	25	0	2	0	1	0
80	4 YR	2	4	1	2	0	2	0	0	0	0	0	0	0	0	0	26	1	1	0	1	0
81	6YRS	2	4	1	2	0	2	0	0	0	0	0	0	0	0	0	20	1	1	0	0	1

CF6MON	FPF1YR	ENERGY	PRTN	MICRONT	TERM_PT	NBW_LBW	WA	HA	W_H	BMImre5yrs	BMIless5yrs	BMINEW	MUAC	Hb	ANEMIAHb	PS	MCV	MCH	MCHC	RDW	ANEMIARCI
1	1	2	2	0	1	2	1	0	1		2	2	0	11.1	0	3	74	25	33	12.3	1
1	1	2	2	0	1	1	0	1	0		2	2	1	10.3	1	1	74	24	33	12.4	1
1	1	2	2	0	1	1	0	0	0		3	3	1	10.3	1	3	74	24	33	12.4	1
0	1	2	2	0	1	1	2	1	2	2		2		11.4	1	3	78	26	34	12.4	0
1	1	2	2	0	1	1	2	1	2	2		2		12.4	0	3	80	27	33	11	0
1	1	1	1	0	1	2	3	2	2		2	2	3	16.9	0p	3	93	25	30	14.2	0
1	1	2	2	0	1	1	2	1	1	2		2		17	0p	3	81	26	32	13.6	0
1	1	2	2	0	1	2	1	1	0	2		2		13.6	0	3	75	25	33	12.4	0
1	1	2	2	0	1	1	0	0	1	2		2		12.5	0	3	86	28	33	11.7	0
1	1	2	2	0	1	1	2	0	0	2		2		11.4	1	3	76	23	33	15.1	0
1	1	2	2	0	1	1	1	0	1	2		2		11.4	1	3	85	28	34	12.7	0
0	1	2	2	0	1	1	0	0	1	2		2		12.4	0	3	85	29	34	12.6	0
1	1	2	2	0	1	1	2	1	2		2	2	1	17.5	0p	3	81	26	32	13.6	0
1	1	2	2	0	1	2	0	1	0		3	3	0	17	0p	3	87	28	33	13.6	0
1	1	2	2	0	1	1	2	0	1	1		1		12.8	0	3	82	27	33	13.1	0
1	1	2	2	0	1	1	1	0	1	3		3		12	0	3	87	28	33	13.3	0
0	1	2	2	0	1	1	2	1	2	2		2		11.5	1	3	82	27	33	13.1	0
0	1	2	2	0	1	1	1	0	1		2	2	0	12.8	0	3	77	25	33	11.9	0
1	1	2	2	0	1	1	0	0	1	3		3		13.1	0	3	80	28	35	10.4	0
1	0	2	2	0	1	1	2	0	3	3		3		12.8	0	3	77	25	33	12.3	0
0	1	2	2	0	1	1	1	0	1	2		2		13.4	0	3	78	25	33	12.8	0
1	1	2	2	0	1	1	3	2	1	2		2		15	0	3	82	28	34	12.7	0
1	1	2	2	0	1	1	2	0	2	2		2		11.2	1	3	77	24	31	14.4	0

1	0	2	2	0	1	1	0	1	0	2		2		11.6	0	3	85	28	32	12.5	0
1	1	2	2	0	1	1	0	0	1		3	3	0	12	0	3	82	29	32	12.6	0
1	1	2	2	0	1	1	2	0	2	1		1		19	0p	3	73	23	32	16.2	1
0	1	2	2	0	1	2	1	1	2		1	1	1	22.4	0p	3	73	23	32	16.3	1
1	1	2	2	0	1	1	2	1	2	1		1		11.8	0	3	76	26	34	12.5	0
1	1	2	2	0	1	1	1	0	1	2		2		13	0	3	82	27	33	11.7	0
1	1	2	2	0	1	2	0	0	1	3		3		13.4	0	3	87	30	34	12.5	0
0	1	2	2	0	1	1	3	2	3	1		1		13.1	0	3	81	27	34	12.1	0
1	1	1	1	0	1	1	0	0	0	3		3		13.6	0	3	82	27	33	12.7	0
1	0	2	2	0	1	1	3	1	2	3		3		11.8	0	3	85	29	34	12.7	0
1	1	2	2	0	2	2	0	0	1	2		2		12.1	0	3	75	24	32	13.4	1
1	1	2	2	0	1	1	2	1	1	3		3		13.2	0	3	77	25	33	12.3	0
1	1	2	2	0	2	2	0	0	1	2		2		13.2	0	3	78	26	33	13.5	0
1	1	2	2	0	1	1	3	1	3	2		2		12.1	0	3	82	26	32	12.9	0
1	1	2	2	0	1	2	2	0	3	2		2		12.8	0	3	87	29	33	12.5	0
0	1	2	2	0	1	1	2	1	2	2		2		12.6	0	3	79	25	33	12.7	0
0	1	2	2	0	1	2	3	2	1	2		2		14.2	0	3	82	26	32	14.5	0
1	1	2	2	0	1	1	3	2	2	1		1		18.8	0p	3	83	29	35	11.7	0
0	1	1	1	0	1	1	3	1	2	1		1		11.8	0	3	83	26	31	12.5	0
1	1	2	2	0	1	1	1	0	1	2		2		13.2	0	3	84	27	33	12.6	0
1	1	2	2	0	1	1	2	1	1	1		1		17	0p	3	77	26	34	13.5	0
1	1	2	2	0	1	1	2	2	1	2		2		12.3	0	3	82	27	32	14.3	0
1	1	3	3	0	1	1	0	0	0	5		3		11.7	0	1	73	23	32	13.9	1
0	1	2	2	0	1	1	2	0	1	2		2		10.3	1	1	80	25	30	11.5	1
1	1	1	1	0	2	1	3	3	2	1		1		17	0p	3	86	29	34	12.7	0

0	1	2	2	0	1	1	0	0	0	3		3		12.1	0	3	83	28	33	14.1	0
1	1	2	2	0	1	1	0	0	1	2		2		11.6	0	3	80	28	35	13.5	0
1	1	2	2	0	1	1	2	2	1	1		1		11.4	1	3	80	27	34	11.8	0
0	1	2	2	0	1	1	1	0	2	2		2		13.8	0	3	87	29	34	12.3	0
1	1	2	2	0	1	1	1	1	1	2		2		14	0	3	86	29	34	13.6	0
1	1	2	2	0	1	1	1	0	1		2	2	1	12.1	0	3	75	25	33	12.6	0
1	1	2	2	0	1	1	2	1	3	1		1		12.6	0	3	83	28	34.9	11.5	0
1	0	2	2	0	1	1	2	1	2		2	2	2	12.3	0	3	80	26	33	13.2	0
1	1	2	2	0	1	2	2	3	2		1	1	1	17	0p	1	81	27	33	16.4	0
1	1	2	2	0	1	1	0	0	0		2	2	0	11.8	0	3	86	28	33	12.4	0
0	1	2	2	0	1	1	1	0	2		2	2	0	10.3	1	1	73	23	32	16	1
1	1	2	2	0	1	1	1	1	0		2	2	1	11.6	0	3	84	28	33	12.5	0
1	0	2	2	0	1	1	3	3	3		1	1	3	11.8	0	3	86	28	33	12.8	0
0	1	2	2	0	1	1	1	1	1		2	2	0	13.6	0	3	71	23	32	15.3	0
0	1	2	2	0	1	1	3	3	1	2		2		17.7	0p	3	64	18	29	18.8	0
1	1	2	2	0	1	1	0	2	1		2	2	1	9.1	2	1	71	23	30	13.2	1
0	1	2	2	0	2	1	2	2	3		1	1	2	10.5	1	1	64	20	31	16.8	1
1	1	2	2	1	1	1	0	0	0		3	3	0	11.7	0	3	74	26	34	13.1	0
1	1	2	2	0	1	1	1	1	1		2	2	0	11.1	0	3	73	25	35	13.1	0
1	1	2	2	0	1	1	1	1	1		2	2	0	12.3	0	3	80	29	36	12.3	0
1	1	2	2	0	2	2	1	1	2		2	2	0	10.6	1	1	70	22	31	16.9	1
0	1	2	2	0	1	1	2	2	1		2	2	0	10.6	1	1	72	21	29	15	1
1	1	2	2	0	1	1	0	0	1		3	3	0	11.3	0	3	82	28	33	12.1	0
1	0	2	2	0	1	1	0	3	1		2	2	0	10.1	1	1	69	23	33	14.4	1
1	1	2	2	0	1	2	1	1	1		2	2	0	16.9	0p	3	73	23	32	15	0

0	1	2	2	0	1	1	3	3	3		1	1	0	10.8	1	1	73	22	33	14	1
1	1	2	2	0	1	2	2	1	3		2	2	1	18.8	0p	3	78	25	32	14.9	0
1	1	2	2	0	1	1	4	3	2		1	1	3	18	0p	1	63	19	30	17.6	1
1	1	2	2	0	1	1	1	1	2	2		2		12.9	0	3	83	28	34	12.7	0
1	1	2	2	0	1	1	3	0	3	1		1		13.2	0	3	85	28	32	13.4	0
1	1	2	2	0	1	1	1	0	2		2	2	0	12.6	0	3	88	29	33	12.6	0
0	1	2	2	0	1	1	1	2	0	2		2		8.6	2	1	66	18	28	18	1